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Physical diagnosis : with case examples

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PHYSICAL DIAGNOSIS

PHYSICAL DIAGNOSIS

WITH CASE EXAMPLES OF THE INDUCTIVE METHOD

BY

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*WITH EIGHTY-EIGHT ILLUSTRATIONS IN THE TEXT
AND THIRTY-TWO PLATES*

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PREFACE

In these days of extensive, almost entangling, and yet generally exact laboratory training in the methods and processes of diagnosis, there is a tendency among medical students to slight and become superficial in the responsibility and cultivation of careful and minute clinical observation: students are more apt to scrutinize the chemical and microscopic aspects of a patient's secretions and excretions than to perceive and analyze his physical signs and objective symptoms. With a clear appreciation of the accuracy and utility of laboratory results in their wonderful details and penetrating ramifications, it behooves us, nevertheless, to enlarge upon and emphasize, in the following pages, the physical methods of observation of the individual's bodily organs and their functions.

In my teaching experience it has appeared that the average medical student tends to neglect or misapprehend the intrinsic importance of two things, namely: technic and reason. He is inclined to exhibit a superficial and hasty zeal and endeavor to get at the physical signs somehow, but without methodic and patient procedure and practise; and his mental drift is toward jumping at conclusions from insufficient data and inefficient correlation.

It is the double-barreled purpose of this book, its dual *raison d'être*, to counteract, as far as text-book influence may (of course, less potent than the personal power of the teacher), this deficiency and tendency in the student body of our medical colleges.

First, insistence and emphasis is to be placed upon technical practise and precision; upon those principles of action and methods of procedure, concentrated and painstaking attention to which will prevent meager and confused results in the interpretation of the signs thus elicited.

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Secondly, the inculcation of the inductive habit of thinking: the analysis of observed facts; the perception of their similarity of meaning; and thus the avoidance of an inconsequential, fallacious empiricism. We need in clinical work more of the line of diagnostic thinking exemplified by Dr. (Sir) Arthur Conan Doyle's immortal consulting detective, namely, the "synthetic induction" habit; indeed, it is the author's custom to recommend to medical students the careful reading of the "Adventures and Memoirs of Sherlock Holmes." It is in the province of the physician to discover the evidences (signs) of disease as it was his to detect those of crime; to deduce the meaning and identity of the signs (the logic of synthesis); then to collect and correlate these separate deductions and to perceive their similarity in harmony with a single conclusion inferred from them (the logic of induction).

It will be noticed, however, that the application of the inductive method is limited to the sections on the special physical diagnosis of diseases of the lungs and heart; to the citation and analysis of certain individual cases illustrating the method; it would, obviously, be irrelevant and irrational to force the inductive treatment of the subject throughout the work, particularly at the beginning, where the elemental facts and principles and the description and details of technic must first be apprehended in order to make the method productive.

It is the spirit, the trend of the inductive method which is adopted here. The purpose is not to be cyclopedic in the mere enumeration of physical signs to burden the memory: the technical, logical, and practical are more worthy of emphasis and development. It is vitally important that the student, in the practise of a careful technic, gains assurance of observation and intelligence of apperception; and that he learns to think logically upon what he thus finds.

It is hoped that this book may accord, in some measure, with the ideals, standards, and rapidly rising requirements of modern training in our medical schools of all sizes, rank of attainment, prestige, and affiliation. We believe that, at the present time, the majority of students graduated from our medical colleges fall far short of an adequate knowledge of the facts, principles, and clinical importance of physical diagnosis, and of practical training in the technic of the methods of physical examination.

Furthermore, in view of the increasing number—already considerable—of medical examiners for life-insurance companies, and of physical directors for college, club, and Y. M. C. A. gymnasiums, what has just been indicated in the previous paragraph calls for serious, energetic, and enthusiastic reflection and regeneration. Besides, it may not be questioned that a more thorough teaching of physical diagnosis, in its essentials at least, should be given in our dental college curricula; dental students should be required to learn especially those physical signs of the heart which so constantly sustain vital relations to the precautions, complications, and progress of anesthesia.

Other works and the latest literature bearing upon the subject have been drawn upon for many of the long-established and lately demonstrated facts tested and proven by competent experience.

As to the illustrations, most of which were borrowed because of their suitability to the text, the author wishes to express, however feebly, his especial appreciation and admiration of those loaned by Dr. Glentworth R. Butler with the kindest courtesy possible.

Finally, to Dr. Pfahler I am highly indebted for his valuable chapter on Röntgenography, and for the use of his very fine and rare and complete Röntgenograms. It is a pleasure to commend his technical ingenuity and skill not only, but his penetrating and judicious clinical interpretations as well.

H. S. A.

1836 Wallace Street, Philadelphia.

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INTRODUCTION

INTRODUCTION

Definition.—Practically, *physical diagnosis* is the art of *detecting* and *discriminating* diseases of the *chest* and *abdomen* by the aid of *signs* denoting the abnormal *physical* conditions of the organs or structures under examination.

Broadly speaking, it is the *objective investigation* of the whole body by the use of the physician's special senses, especially of sight, of touch, and of hearing, with the appropriate aid of instruments of precision; so that the perceived and demonstrable results of the physical examination, in the *physical signs* elicited, may, severally or conjointly, be said to be *visible*, *palpable*, and *audible*. In the scientific development of medical practise, however, the methods of physical diagnosis are recognized as most applicable to the determination of the anatomic and physiologic deviations caused by affections of the thoracic and abdominal viscera: thus, of the chest, the heart and lungs are examined and studied; of the belly, the liver, spleen, stomach, bowels, and kidneys. As, early in the century just past, Piorry, in France, expressed it in his word "organography," the idea of physical diagnosis is embraced fairly well in the determination of the actual and relative position, material condition, and sensible functional action of the organs contained within the body.

Standpoint and Importance.—To the superficial comprehensiveness of the "physiognomy of disease" (Gairdner) physical diagnosis adds penetration and precision of method, and rational reliability of evidence; the value of the objective symptoms of the former being controlled and tested, augmented or invalidated, by the physical signs of the latter. Thus, while such external phenomena as refer to the general appearance of the patient may indicate as a guide-post the internal organ or organs in which disease is to be looked for, it is only as the methods of physical examination are carefully applied that we really learn the presence, character, extent, and stage of an organic pulmonary or cardiac affection. For instance, we may note the stature, build, posture, complexion, and facial expression, apparent age as compared with the real age, finger-ends, dropsy, dyspnea, temperature, cough, etc., pointing to thoracic disease, in the line of physiognomic diagnosis. When the intimate association of the respiratory and circulatory functions is realized, however, obviously it is impossible to define with any precision whether the heart or a lung is the seat of disease, what that disease is, what it involves, and whether or not both systems are affected independently or sequentially, unless the more exact results of systematic physical exploration are sought for and explained.

The importance, then, of physical diagnosis, in diseases of the chest especially, exists primarily and essentially in the fact that it puts forth physical evidence of morbid changes and pathologic processes which can be learned in no other way than by the trained eye, ear, and hand of the responsible physician; evidence gotten independently of the sometimes misleading and malingered statements of the patient's medical history or symptoms; and secondarily and indirectly because it not only serves as a check or control over the significance of the complexus of mere symptoms of thoracic affections, but because it contributes such positive and accurate evidence as to enhance the value of a rational symptomatology and give completeness to the general medical diagnosis: our whole clinical conception of a given case is rendered clearer, deeper, and broader in the related facts of its etiology, pathologic anatomy, and semeiology; and by so much prepares for and sustains a wiser prognosis and safer and more successful management.

Physical Signs.—These have been referred to following the definition of physical diagnosis, and it is simply purposed here to describe more definitely their general character and meaning. It should be borne in mind that while purely objective manifestations, physical signs are not to be confused with objective symptoms, so-called; particularly as the latter are not infrequently spoken of as "signs" of disease. A physical sign is always objective; but an objective phenomenon is not necessarily or always a physical sign, for it may be only an objective symptom. To illustrate: rapidity of respiration and a one-sided bulging of the chest are objectively perceived; but the former is a symptom discovered by the general observation, while the latter is a physical sign observed by the special, comparative, and critical method of inspection. Again, breathlessness (dyspnea), a bluish pallor of the lips and cheeks (cyanosis), are objective symptoms or signs; while an irregular, turbulent throbbing over the heart and an abnormal position of the apex-beat (palpable) and weak, arhythmic sounds (audible) are physical signs elicited by the special methods of palpation and auscultation. It should be noted, too, that subjective symptoms, or those felt only by the patient, as pain, vertigo, are not all exclusively so; for example, coughing and shortness of breath are both subjective-objective symptoms, recognized by the physician as well as known to the patient.

It is literally true, then, that physical signs are special signs, (1) because recognized objectively by the three special senses of the physician—vision, feeling, and hearing; (2) because they are the result of the application of special methods of physical examination. They are physical signs because they represent changes in the physical properties of organs consequent upon certain anatomic conditions and changes. They are thus present in health as well as in disease; and, logically, they must be known in states of health before they can be the means of aiding us in distinguishing diseased conditions from normal conditions; and further, it must be known what structural changes they evidence in the various affections, in order that these may be differentiated. "Symptoms guide us, generally, by physiological inference; physical signs, by anatomical necessity" (Hartshorne). Our knowledge of the significance and value of physical signs is, of course, the outcome of close, extended, and multitudinous observations, confirmed by examinations of the morbid changes discovered *post mortem*; and by clinical pathology (pathologic physiology).

Physical Diagnosis as a Science.—Jevons says that "a science teaches us to know, and an art to do." Not only has physical diagnosis gradually acquired for itself this positive, precise, and practical importance of a special art, but it is also, if not an exact science, at least largely and signally scientific. Ophthalmology has been called the most mathematically accurate of the specialties of medical practise; physical diagnosis, the mathematics of internal medicine; not because of any marked numerical certainty, but because of its accuracy of method and logical soundness and synthetic probability of results. For science is not only the "observing, recording, verifying, and formulating of facts"; it is also the recognition and explanation of the relations of facts, and the principles governing them. An art always presupposes a certain amount of science; and it is the province of the science of physical diagnosis to analyze, collate, classify, and systematize into an organic whole the known physical signs; to perceive their characteristics, to determine their causes, to interpret their significance.

The fact that the art of physical diagnosis depends upon (under next heading) a preliminary knowledge of physics, anatomy, physiology, pathology—sciences—makes it fundamentally and truly scientific. And eminently true is it that the method of this practise is based upon the general method of inductive logic, the logic of science, involving, as it does, not simply the collection of facts, but their verification and explanation by means of the contributory processes of comparative observations and separate deductions.

Relations of Physical Diagnosis.—Physical diagnosis is scientifically the eldest, as *clinical microscopy* and *clinical chemistry* are the younger brothers in the working out of modern medical diagnosis; worthy children of a worthy parent—general semeiology: neither, alone, sufficient without the others, nor two combined without the other, but the precise and special results of the younger clarifying and controlling, or confirming, and reciprocally being guided and valued by the broad and rational basis of the eldest.

Physical diagnosis is directly dependent, however, upon *five* (5) *fundamental sciences,* the thorough study and adequate knowledge of which are essential to any accuracy of physical exploration and soundness of diagnostic deduction. These are as follows: (1) *Medical physics*; (2) *anatomy* (especially *topographic* and *relational*); (3) *physiology* (including the experimental); (4) *pathology* (especially the gross morbid changes in structure); (5) *logic.*

(1) The very phrase and term, physical diagnosis (title, physician), presupposes a distinct cognizance of the phenomena and principles of physics as applied to medical diagnosis. For a consistent apprehension of the significance of most of the important physical signs elicited by feeling of the chest walls (palpation), sounding (percussion), and direct or instrumental—stethoscopic—listening (auscultation), there must be some clear knowledge of, for example, the attributes of sounds, such as intensity, pitch, quality; the changes and various effects in their production, conduction, and dissipation in and over different structures and organs, such as pertain to air-containing and airless organs, bronchial and intestinal tubes; the acoustic effects of increased density, as of a morbid deposit in the lungs, of diminished elasticity, of intervening normal or pathologic structures, and of the presence abnormally of gaseous, liquid, and solid substances upon the character and propagation of sound waves; the relations of the volume and tension of membranous organs or cavities to the pitch, especially, of percussion vibrations; and the

dependence of the phenomena of the cardiovascular system upon the principles of hydrodynamics (hemodynamics?).

(2) That an accurate acquaintance with at least gross anatomy is a *sine qua non* in the study and practise of physical diagnosis needs but to be mentioned to meet with ready assent. For how can the location, size, shape, and mobility of outline of physically demonstrable organs be determined, with their pathologic changes, unless their anatomic relations to surface landmarks, as well as to each other, are clearly known? "The ambition of the student of anatomy is, or should be, the ability to see through the body, perceiving in the mind's eye all the structures included therein, and their complicated relations to each other. This ability the student must gain somehow, if he is to achieve real success in medicine or surgery; for upon this knowledge are based both physical diagnosis and surgical procedure" (Jackson: University of Missouri), *Journal Amer. Med. Assn.*, September 21, 1901. It should be borne in mind, too, that a knowledge of regional anatomy is fundamental to the precise application of practically all of the methods of physical investigation, and especially of percussion, simple or auscultatory.

(3) As, in physical diagnosis, we are dealing with living, functioning organs, though presumably diseased ones, it is plain that physiology is likewise a basal branch. The normal, physiologic functions of the heart and lungs give rise to physical signs (normal), which must be perceived and understood before the abnormal changes can be recognized and interpreted. A familiar, ready, and, as it were, standard acquaintance with the pulmonary and cardiac movements and cycles and their sounds, as met with in health, is an indispensable preparation to an adequate appreciation of abnormal variations and conditions. While there is a large field for post-graduate original work in the domain of experimental physiology, especially in the study of cardiovascular phenomena, at the same time the facts and laws already established constitute an important part of the preliminary knowledge in physical diagnosis.

(4) Following naturally the preceding requirement is the very practical necessity of knowing not only independently the physical characteristics of the gross morbid organic changes, but also, and particularly, their more or less definite relations to the abnormal physical signs observed and elicited during life: a knowledge of the *ante-mortem* physical phenomena should be compared with that of the *post-mortem* findings. Modifications of the signs as affected by the various stages of certain pathologic processes, as in pulmonary

tuberculosis, lobar pneumonitis, pericarditis, etc., it is also essential to apprehend. Neither can the facts and laws of physics and experimental physiology be applied to the explanation of the phenomena of pulmonary and cardiac diseases—and their treatment, also (Balfour) —unless the pathology or mode of their production and the consequences of their action upon the functions of other and related organs are understood.

(5) Logic is the fundamental science of all the sciences (*scientia scientiarum*); the physiology of thought; and in no part of medical practise is the logical training and habit of mind more truly necessary and responsible than in diagnosis. Physical diagnosis and logic are analogous and coterminous in that they aim to apply clear methods, to understand and prove the evidence, to prevent error and fallacy, and to attain a knowledge of the nature of the results—their significance. Not only upon alert and accurate observation does a diagnosis rest, but much more upon normal, methodical, right reasoning: the signs must signify, the data must be explained by means of logical inference. It is "with the meaning side of ideas that logic has to do" (Creighton: Cornell University). Physical diagnosis consists mainly of a series of deductions from a number of "well-established generalities" (Bain) yielded by the applicable sciences referred to in the paragraphs just preceding. No better or more common example can be given of the deductive method (by syllogism) than in the case of the diagnosis of simple valvular heart-murmurs by auscultation.

So much for the prerequisites to the study of physical diagnosis. On the other hand, *prognosis* and *treatment* are dependent upon physical diagnosis, in large measure, for the important objective evidence it furnishes in pulmonary and cardiac affections especially. For instance, in lobar pneumonia the physical signs indicating extension of congestion and consolidation, or of delayed resolution, or of diminution of the accentuation of the pulmonary valve sound, are of particular value in estimating the probable course and complications. and in deciding rationally and promptly upon the proper management of the patient. Again, the changed character of a cardiac murmur and first, systolic, or ventricular sound, the altered position of the apex-beat, and lateral extension of percussion dulness over the heart indicative of its more or less acute dilation, likewise present opportune and important hints in these respects. Similarly, in other diseases, as in the acute infectious fevers, the prognosis and treatment often require modification and extra cautiousness of judgment and exercise of skill because of the thoracic complications revealed by the methods of physical examination.

Qualifications for the Study of Physical Diagnosis.—Back of the special knowledge and training acquired in the fundamentals lie the advantages of a degree of general equipment that cannot fail to be of marked and far-reaching practical benefit. This is twofold: *physical* and *psychic.* These are necessary for the work to be done in the corresponding and cooperating processes of observation and explanation. Since the results of physical examination depend entirely and exclusively upon the perceptive and ratiocinative sense-organs and faculties of the physician, the finer and stronger these are innate, and the better developed and balanced their powers by previous education, the readier, broader, and sounder the diagnosis arrived at.

This means that anatomic and functional deficiencies of eye, ear, and hand may prove to be serious handicaps in clinical work. Very poor eyesight from exaggerated refractive and asymmetric ocular troubles; partial deafness in one or both ears or from perforated or destroyed tympanic membranes; and nerve degenerations, finger losses, deformities, or disabilities from disease, accident, or operation, however slight, are more or less drawbacks to efficiency.

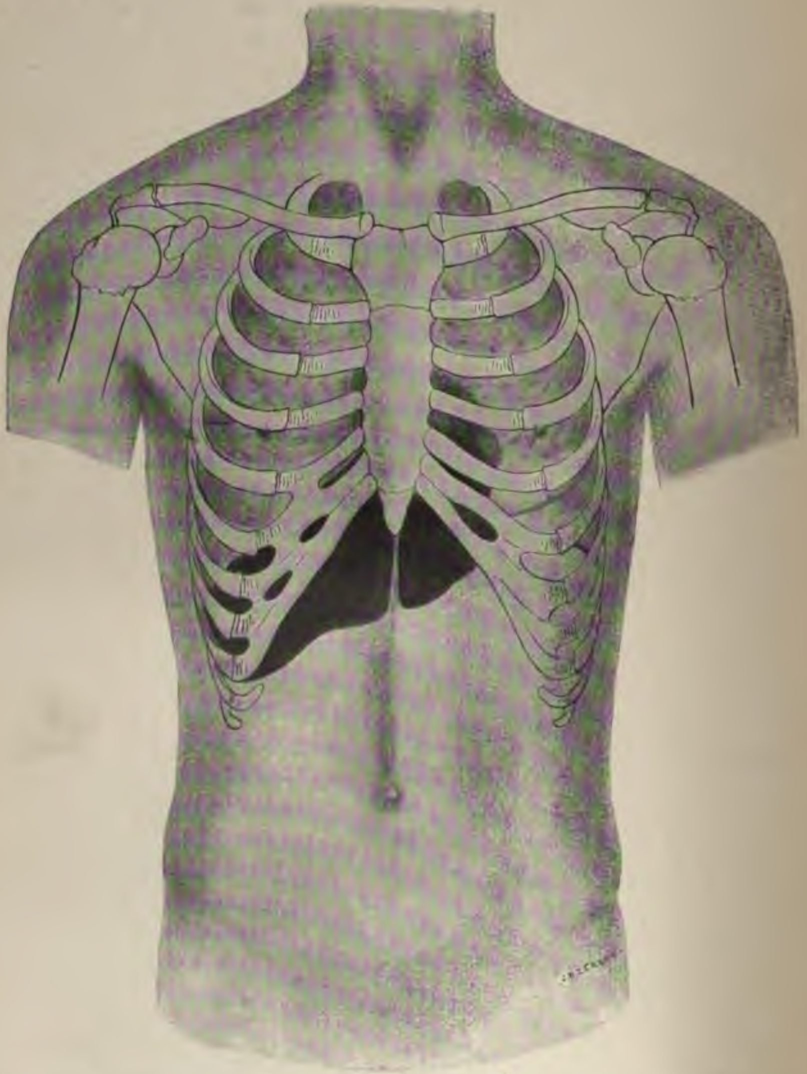
Of course, even with physical wholeness and robustness, naturally some are more talented, have keener sense-perceptions than others. Nevertheless, though not every one can inherit, cultivate, and manifest the exquisite acumen and precision of a Flint or Osler, a DaCosta or Loomis or Pepper, the personal equation of every one can be wondrously improved by attentive, earnest, patient, persistent, thoroughgoing practise. To this end the student will realize that previous manual training and habits of accurate observation in the study of the natural sciences, such as zoology, biology, mineralogy, botany, and chemistry, are invaluable requisites. In physical diagnosis, just as in the generalizations from the phenomena of natural objects, organisms, and experiments, we essay to *observe closely by comparison*—especially in the detection of abnormalities and asymmetries—to analyze or relate, and to classify and interpret. Obviously, the perceptive powers cultivated in field and laboratory, the technical skill acquired in handling instruments of precision, and systematic procedures thereby inculcated and ingrained are hardly less than absolutely necessary modern conditions of intellectual preparedness for successful results in this most objective department of general medical diagnosis.

The benefits from the study and practise of physical diagnosis bear the same sequential relation to professional progress in diagnostic skill and inference that those do to preparatory education which are developed and acquired in attaining the qualifications for the study of physical diagnosis. The eye, ear, and hand become more highly sensitive and specialized, the prompt concentration and circumscription of attention are sharpened, and the inferences derived from reasoning upon the results of observation are facilitated, and their consistency and probability enhanced. *A fortiori,* the work and reliability of medical diagnosis in general are furthered and established by so much: perspicacity becomes, as it were, a professional habit at all times.

PART I

THE CHEST (THORAX)

PLATE I



Anterior Aspect. Shows the Normal Position and Relations of the Lungs and Heart to Each Other and to the Bony Thorax.

SECTION I

METHODS AND TECHNIC: PHYSICAL SIGNS AND DIAGNOSIS IN GENERAL

CHAPTER I

TOPOGRAPHIC AND RELATIONAL ANATOMY OF THE CHEST

NEXT to the actual detection of physical signs, the most important diagnostic point is their anatomic localization. However, as some knowledge of the surface anatomy in relation to the underlying organs is fundamental to the development of the methods of physical examination, the topography of the chest will be considered first. It is assumed that the student is tolerably familiar with the descriptive anatomy of the heart and lungs, and with such elemental physical characteristics as their situation, size, shape, density, contiguity, etc. (See Introduction.)

For localizing physical signs as precisely as possible we utilize partly the more or less obvious anatomic spaces and prominences (landmarks); and partly, local regions outlined arbitrarily, and yet as naturally and conveniently as may be, by the use of certain lines conceived as drawn upon the surface of the chest. It is of great importance, in general as well as in physical diagnosis, that an accurate knowledge of thoracic landmarks be obtained, as it assures accuracy in locating the diseased parts or organs by the principal means—i. e., the surface indications.

Such knowledge is also necessary in any intelligent description of the site of lesions, whether in recitation, clinical conference or consultation, discussion or written article; and likewise in describing and recording changes of position, and of extent and direction of the boundaries of organs. This holds true whether the description refers merely to an approximate designation, as of a region, or to the more accurate pointing to a numbered rib or interspace, or measured relation to another recognized anatomical prominence or arbitrary line.

ANATOMIC LANDMARKS OF THE CHEST

(1) **Clavicle.**—Starting on the front of the thorax, from above, are these double-curved bones, the clavicles, prominent dividing lines between the neck and chest (and supra- and infraclavicular regions), affording fixation points for measurements, and covering the first rib on either side.

(2) **Sternum.**—At the top of the breast-bone is the *suprasternal notch,* on a level with the disk between the second and third dorsal vertebræ.

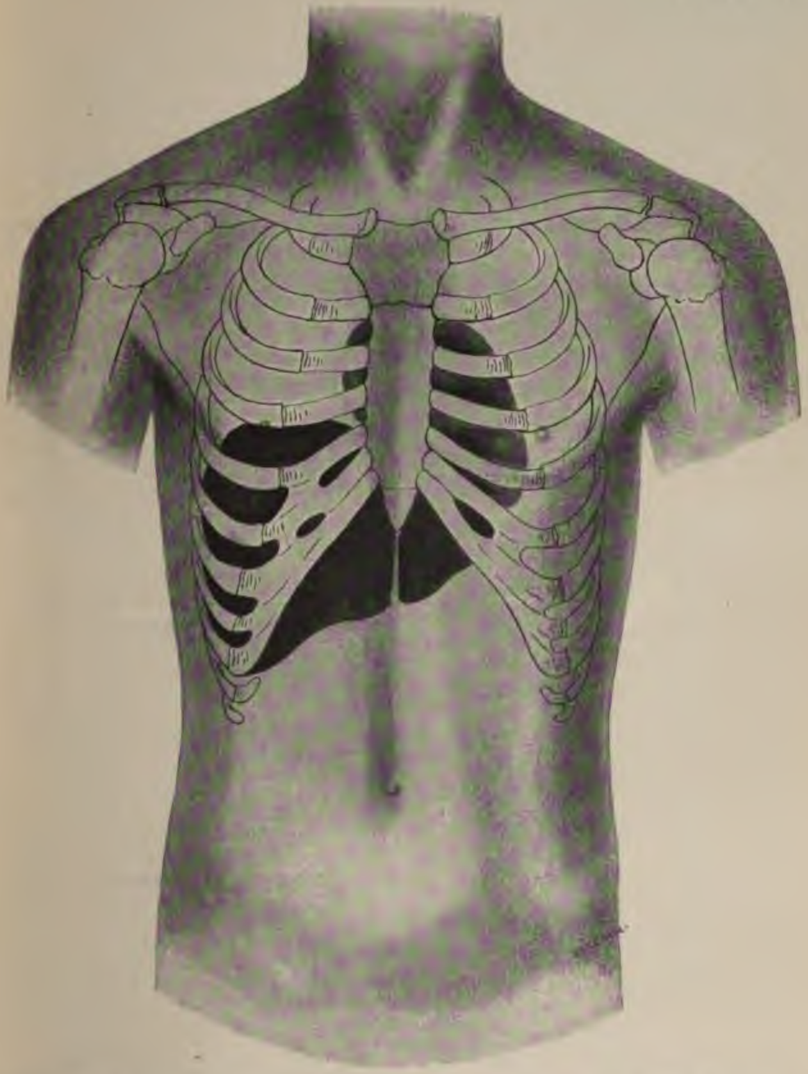
Passing downward from the notch, about one and one-half inches below, is a transverse ridge or projection, slight but distinct, the *angle of Louis* (Angulus Ludovici). It is formed by the junction of the manubrium with the body of the sternum, is practically always to be seen and felt in every individual (less so in females and in the obese), and affords an infallible guide in the counting of ribs, as it corresponds with the middle of the cartilage of the second rib. It is also on a plane with the body of the fifth dorsal vertebra.

At the lower end of the sternum is the slight depression caused by the dipping backward (variable) of the ensiform or xiphoid appendix below its junction with the body (gladiolus) of the sternum. Sometimes it curves and projects forward so that its tip forms a small nodule, or nipple-like eminence. The sternoxiphoid junction is nearly on a line with the disk between the ninth and tenth dorsal vertebræ.

(3) **Ribs and Intercostal Spaces.**—METHOD OF COUNTING RIBS.—Ribs are the most commonly used bony landmarks in all physical examinations of the chest. To know them and count them, therefore, requires constant practise, so that ready familiarity in localization may be attained.

As pointed out above, the angle of Louis is always a reliable starting-point, the finger running along horizontally over the second rib on either side, the number of any particular rib being then determined by counting downward and feeling somewhat obliquely outward at the same time. Any desired rib may be followed thus into the axilla, and backward and slightly upward posteriorly toward the spine.

As noted by Holden, the fifth rib corresponds in direction to the lower external border of the pectoralis major.



Anterior Aspect. Shows the Cardiac Relations with the Lungs Removed.

Again, a line drawn horizontally from the nipple round the chest cuts the sixth intercostal space midway (midaxillary line) between the sternum and the spine—a useful rule in tapping fluid from the chest.

Furthermore, when the arm is raised horizontally or a little above, the highest visible digitation of the serratus magnus corresponds with the sixth rib. The two digitations below this lie over the seventh and eighth ribs respectively.

The eleventh and twelfth ribs can be felt when the abdominal wall is relaxed, even in short persons. They slope downward and forward outside the erector spinæ muscles.

It is this downward slope which makes the sternal ends of all the ribs lower than their vertebral articulations. Thus, the third chondrosternal junction is on a level with the body of the sixth dorsal vertebra. Below this, to the seventh rib inclusive, by adding four to the number of the rib in front, its relative level with the corresponding costovertebral articulation behind is immediately known; for example, the fifth rib anteriorly corresponds with the level of the ninth rib posteriorly, and so on.

Upon the back, points of height are also determined by rib-counting, though here it is manifestly more difficult, especially in the corpulent.

(*a*) The tips of the vertebral spines may serve as guides, beginning with the vertebra prominens, or seventh cervical, immediately below which the first rib begins; then the second rib, articulating with the second and third dorsal vertebræ, and so on downward, excepting the tenth, eleventh, and twelfth ribs, which articulate with their respective numbered dorsal vertebræ only. A caution is advisable here. Owing to their downward projection, most of the dorsal spinous processes are on a level with the next numbered rib below: thus, the second dorsal spine corresponds with the third rib, and the ninth spine to the tenth rib; the intervening spines and ribs accordingly; the tenth spine, shorter and less inclined, is between the tenth and eleventh ribs; the last two ribs, like the first one, correspond with their respective numbered vertebral spines.

(*b*) The Seventh Rib.—This lies under the lower angle of the scapula in easy positions of the chest, with the arms resting against the chest and the forearms hanging or folded lightly. Hence the ribs may be counted posteriorly from this point of the scapula representing the position of the seventh rib.

(*c*) The twelfth rib may readily be felt behind at its tip especially, in most people, and affords a starting-point for counting the lower ribs upward.

The intercostal spaces lie below their corresponding numbered ribs. The first and second intercostal spaces are readily recognized, one above, the other below, the level of the angle of Louis. The second and third interspaces are usually the widest, and in fact all the interspaces are wider anteriorly than laterally and posteriorly.

(4) **Epigastric Angle.**—This is the angle pointing upward at the xiphoid cartilage, and is formed on its sides by the lower borders of the converging and coalescing lower ribs. It is normally and nearly, in the average person, a right angle during moderate or ordinary breathing; with deeper respiration, the ribs rising and falling, the angle varies, being somewhat more obtuse at the end of a full inspiration, with the ribs more horizontal, and more acute at the end of deep expiration, when the ribs slant downward more.

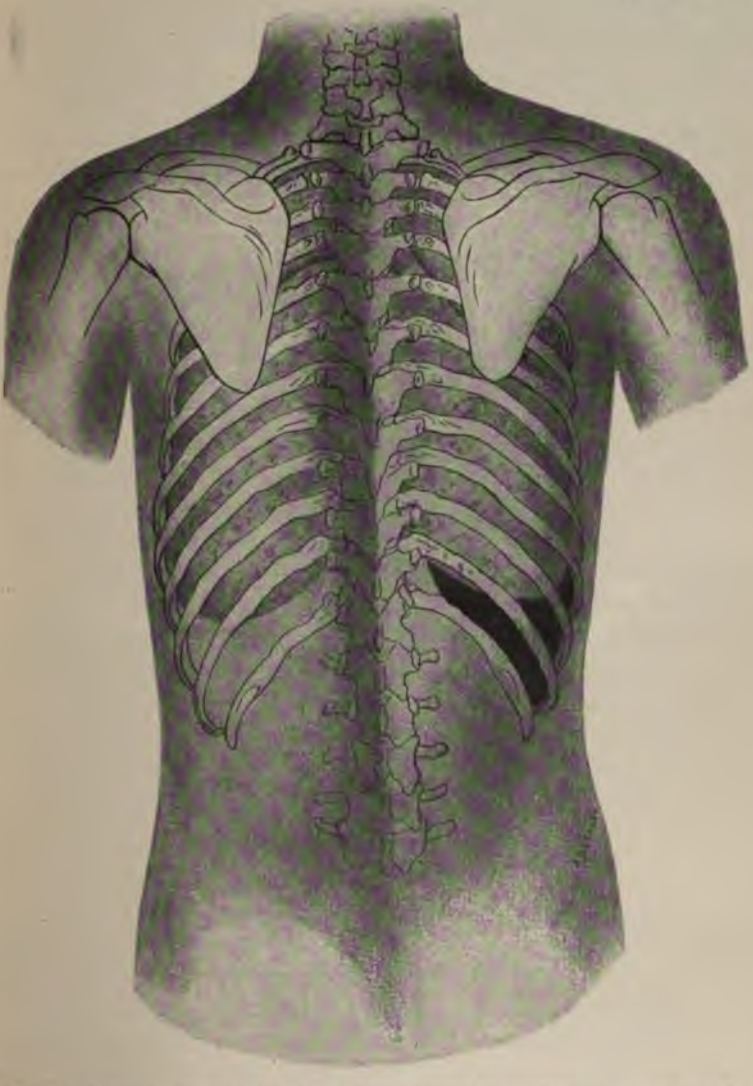
(5) **Nipple.**—This prominent landmark is not a strictly reliable one for constancy and accuracy in localization, especially in females, where the size, shape, and pendulousness of the mammary gland are inevitably and naturally so variable. And, although in the male even its position is not infrequently anomalous, here it is sufficiently constant, in a majority of instances, at least, to be a fairly good point for localization, as it usually lies over the fourth rib or interspace, sometimes over the fifth rib, less commonly over the fifth interspace; or about three-fourths of an inch external to the cartilages, or about four inches from the middle of the sternum.

(6) **Mammary Gland.**—This gland aids in localizing physical signs principally in giving nominal designation to its region, extending horizontally from the sternal border to the anterior boundary of the axilla. Vertically, its circumferential attachment reaches usually from the third to the sixth rib or interspace inclusive.

(7) **Scapula.**—This prominent bone lies on the back from the second to the seventh ribs inclusive. The inner end of the spine of the scapula is on a level with the third dorsal spine, and the inferior scapular angle with the seventh.

(8) **Spine.**—The median groove or furrow is distinct in all. In its middle are the spinous processes, visible by the slight bulgings of the skin over them, except in fat individuals. They may be counted best palpably by the aid of position or friction: either by having the patient bend forward, the convexity of the spine causing the processes to separate and stand out more, or by rubbing briskly up and down

PLATE III



POSTERIOR ASPECT. LANDMARK RELATIONS OF THE LUNGS VIEWED FROM THE BACK.

over them with the finger or towel, their tips then showing bright spots of redness.

The spines of the second, third, fourth, fifth, and sixth cervical vertebræ are scarcely recognizable separately, but the seventh or vertebra prominens, as hinted before, is a distinct landmark for counting and localizing point of vantage. The twelfth dorsal spine may be located also by following in from the twelfth rib, the tip of which may be felt, when the lumbar muscles are relaxed, about three inches from the median furrow.

TOPOGRAPHIC REGIONS OF THE CHEST AND RELATIONAL ANATOMY

The recent tendency to amend the old nomenclature of the divisions of the chest made by certain arbitrary lines conceived as drawn upon its surface, and in which physical signs were to be referred accordingly, is characteristic of progress in method toward a commendable simplicity, desirable accuracy, and judicious flexibility of application. I agree with several recent clinical authors that it is much wiser to refer physical data to bony anatomic landmarks, such as given just previously, using measurements at times; and to divide the chest into regions corresponding only or principally to natural or anatomical outlines. Nevertheless, it so happens that most of the arbitrarily divided regions are really appropriately named on this basis. Hence, and in order that the student may have an intelligent acquaintance with the designated areas as still widely used in medical literature, they are given here with sufficient fulness. The lines auxiliary to a description of these regions are given first.

ARBITRARY LINES AS LANDMARKS

These are to be used rather as guide lines of reference, therefore, and not for a rigid sort of geometric mapping out of the thoracic surface. The natural folds and furrows and fulnesses are simpler and practically sufficient as boundary lines. For determining points of height, however, the clavicle, ribs, and vertebral spines are better than the seven or more imaginary horizontal or latitudinal lines usually drawn. For determining the breadth, certain vertical or longitudinal lines are often useful and commonly used as reference lines in recording and describing physical signs and measurements of localization.

These vertical lines are as follows:

Anteriorly, (1) The *mesosternal* (*midsternal*) line, the middle line of the sternum.
(2) The *sternal* lines, right and left, corresponding to the side margins of the sternum.
(3) The *parasternal* lines, midway between the sternal and the next, or
(4) The *mammillary* lines, passing through the nipples.

Laterally, (5) The *anterior axillary* lines, through the points where the great pectoral muscles leave the chest, when the arms are raised to the horizontal.
(6) The *mesoaxillary* (*midaxillary*) lines, drawn through the middle of the axilla, or midway between the anterior and
(7) The *posterior axillary* lines, running through the points where the latissimus dorsi muscles leave the chest.

Posteriorly, (8) The *scapular* lines, passing through the inferior angles of the scapulæ.
(9) The *mesospinal* (*midspinal*) line, through the vertebral spines.

Of these, by far the most important practically are the mesosternal line, the mammillary line on either side when it coincides with a line passing through the center of the clavicle (*mesoclavicular* or *midclavicular* line), the mesoaxillary lines, and the mesospinal line.

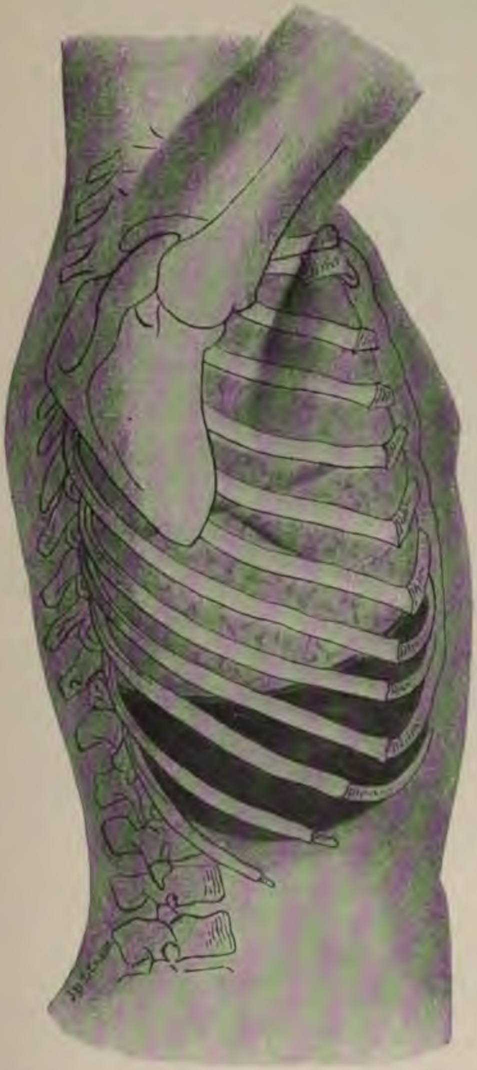
The vertical nipple lines are not always reliable lines of reference because of the variability of the position of the nipple, especially in women, though by no means constant in men; hence, owing to the fixed position of the clavicle, the mesoclavicular line is more dependable, which, in the majority of individuals, nevertheless, does coincide with the mammillary line; therefore the frequent use of the latter term in practise.

REGIONS

ANTERIOR ASPECT

(1) Starting above and in the middle, the first space noticed is the suprasternal depression or region, just above the suprasternal notch, and bounded laterally by the inner borders of the sternomastoid muscles.

PLATE IV



LATERAL ASPECT (RIGHT). TOPOGRAPHIC RELATIONS OF RIGHT LUNG AND LIVER IN THE INFERIOR AXILLARY REGION.

Relational Anatomy.—The suprasternal region is occupied mainly by the trachea; pathologically it may be encroached upon by an aneurismal dilation of the aortic arch.

(2) **Supraclavicular Regions.**—These triangular spaces are directly above the clavicles (inner two-thirds), bounded anteriorly by the sternomastoids and posterolaterally by the trapezius muscle borders.

Relational Anatomy.—Beneath these regions, on either side, are the lung apices (toward the inner thirds of the clavicles), portions of the subclavian and carotid arteries, and of the subclavian and jugular veins. The apex of the lung may rise from one-half to one and three-fourths inches above the upper clavicular border; the left apex is more frequently a little higher than the right. The first rib forms a part of the floor of this region. Just above the inner portion of the clavicle the subclavian artery pulsations may be felt.

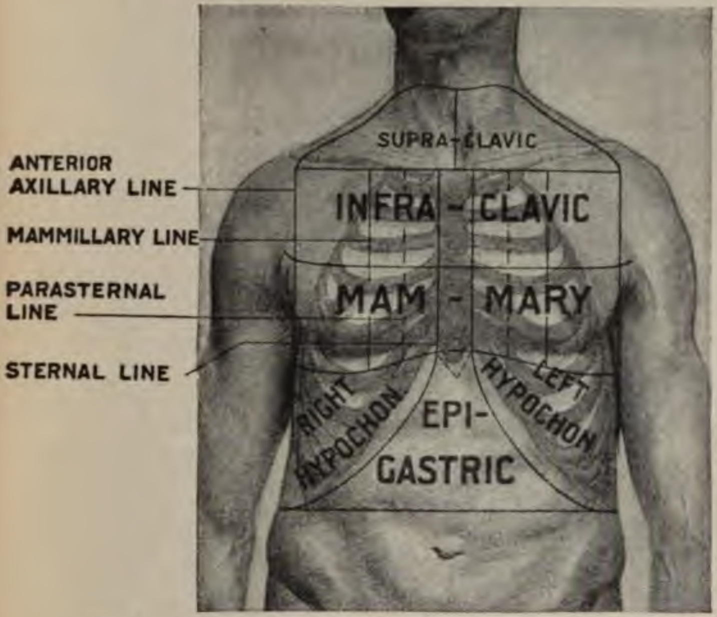


Fig. 1.—Showing the Topographical Areas of the Thorax Anteriorly. (Butler.)

(3) **Clavicular Region.**—This represents practically the margins of the inner two-thirds of the clavicle.

Relational Anatomy.—Behind it is the lung apex on either side. On the right side, at the sternal articulation, lies the bifurcation of the innominate artery and, more externally, the subclavian artery. On the left side, the carotid and subclavian arteries lie more deeply.

(4) **Infraclavicular Regions.**—Situated just below the clavicles, these spaces have no distinct lower boundaries, though somewhat naturally ending where the upper borders of the mammary glands begin—i. e., at the third rib level. Internally they are bounded by the borders of the sternum, and externally by a vertical line passing through the "acromial angle" (formed by the clavicle and the head of the humerus). *Morenheim's depression* is felt in this angle, and is sometimes, though not commonly, used as a reference in the localization of physical signs. *Sibson's furrow,* also, may be used. It represents the lower border of the pectoralis major.

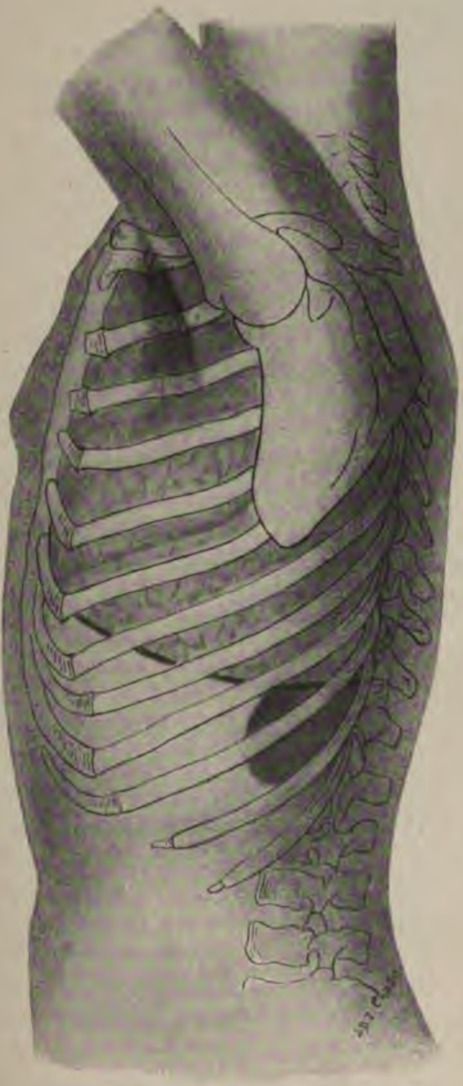
Relational Anatomy.—Besides the upper lobes of the lungs, the infraclavicular fossæ contain the following: on the right side, close to the sternal border, lie the superior vena cava and a part of the aortic arch, while the right bronchus is directly behind the second costal cartilage; on the left side, the pulmonary artery (edge of the sternum), left auricle—second interspace, behind the lung—and left bronchus are situated, the last named a little below the second costal cartilage.

(5) **Mammary Regions.**—Extending from the third to the sixth ribs, as do the glands because of which these areas are named, they are also bounded internally and externally as those preceding. The nipples have already been described as landmarks.

Relational Anatomy.—The contents differ materially on the two sides as related to these regions. On the *right side* are the lung, dome of the liver, right border of the heart, and diaphragm. The lung lies superficially throughout this region, its lower edge, in fact, corresponding to the lower boundary (sixth rib) of the region. The deeply situated right wing of the diaphragm and right lobe of the liver rise to the level of the fourth interspace. The *fissure* between the upper and middle lobes of the lung passes obliquely upward and backward from about the fourth cartilage; that between the middle and lower in the same direction from the fifth interspace. A portion of the right auricle and right ventricle, covered by lung, lie close to the sternum, extending very little from behind its right border, between the third and sixth costal cartilages.

On the *left side* the mammary region is occupied mainly by the heart, partly covered by lung, the anterior edge of which passes obliquely downward and outward from about the level of the fourth

PLATE V



Topographic Relations (Left Lateral Aspect) of the Left Lung and Spleen, Inferior Axillary Region.

cartilage to the fifth rib, thence curving somewhat inward and downward to the sixth rib, within the vertical midclavicular line. The quadrilateral free space thus left is directly over the right ventricle. A portion of the right auricle, and the deeply seated left auricle and ventricle (apex in the fifth interspace inside the midclavicular line), also occupy this region. The anterior end of the *fissure* between the upper and lower lobes of the left lung lies behind the sixth rib in the vertical nipple line.

(6) **Inframammary Regions.**—These naturally extend downward from the sixth ribs to the edges of the false ribs; laterally, from the prolonged outer border of the mammary region to the sternum at the epigastric angle or top of the costal arch.

Relational Anatomy.—On the *right* side, the liver, covered by the diaphragm, and during deep inspiration encroached upon by the lower border of the lung in the sixth or seventh interspaces, are in relation. Normally, the lower border of the liver coincides with the costal edge of the thorax, in the midclavicular line, before reaching obliquely upward and to the left in crossing the median line at a point less than halfway downward between the ensiform cartilage and the navel. On the *left* side, the lower edge of lung extends in the upper and outer portion of this region during full inspiration. The left lobe of the liver occupies the inner part, lying in front of the stomach (cardiac end).

Although the epigastric region lies between the inframammary regions, within the borders of the costal arch, its relational anatomy is properly a part of abdominal topography and physical diagnosis, and will be considered under that head.

(7) **Superior Sternal Region.**—Anatomically, this corresponds to the borders of the manubrium, although most authors extend it below the angle of Louis (the junction landmark with the body of the sternum) to the dividing line between the infraclavicular and mammary regions on a level with the third costal cartilages.

Relational Anatomy.—Here are found the inner edges of the lungs below the level of the second costal cartilages; the bifurcation of the trachea opposite the angle of Louis; the aortic arch (ascending and transverse portions) and pulmonary artery, with their respective valves; the left innominate vein, and a part of the descending vena cava.

(8) **Inferior Sternal Region.**—Corresponding to the remainder of the sternum, this region contains the edge of the right lung descending vertically along the middle line, and at its upper part a

small portion of the left lung; a major part of the right ventricle and origin of the pulmonary artery; a small part of the left ventricle behind, with the origin of the aorta; a part of the right auricle, of the liver, and of the pericardial attachment of the diaphragm.

LATERAL ASPECTS

(9) **Axillary Regions.**—These extend from the points of the axillæ above to the level of the lower borders of the mammary regions below (sixth rib anteriorly); anteriorly and posteriorly by lines drawn through the insertions of the anterior and posterior folds of the axillæ (where the pectoralis major and latissimus dorsi muscles leave the thorax), as seen when the arms are raised sidewise to the horizontal (anterior and posterior axillary lines).

Relational Anatomy.—On both sides are lung tissue, and, deeper, the bronchi and larger bronchial branches.

(10) **Infra-axillary Regions.**—They are bounded above by the axillary regions; below, by the margins of the false ribs; anteriorly, by the external boundary of the inframammary regions; posteriorly, by the infrascapular regions (at the posterior axillary lines).

Relational Anatomy.—On the *right* side, the lung with its lower border sloping downward and backward to the eighth rib in the mid-axillary line, and below, to nearly the edge of the thorax, the liver, are contained. On the *left* side, in addition to the similarly placed lung, are the stomach and spleen.

POSTERIOR ASPECT

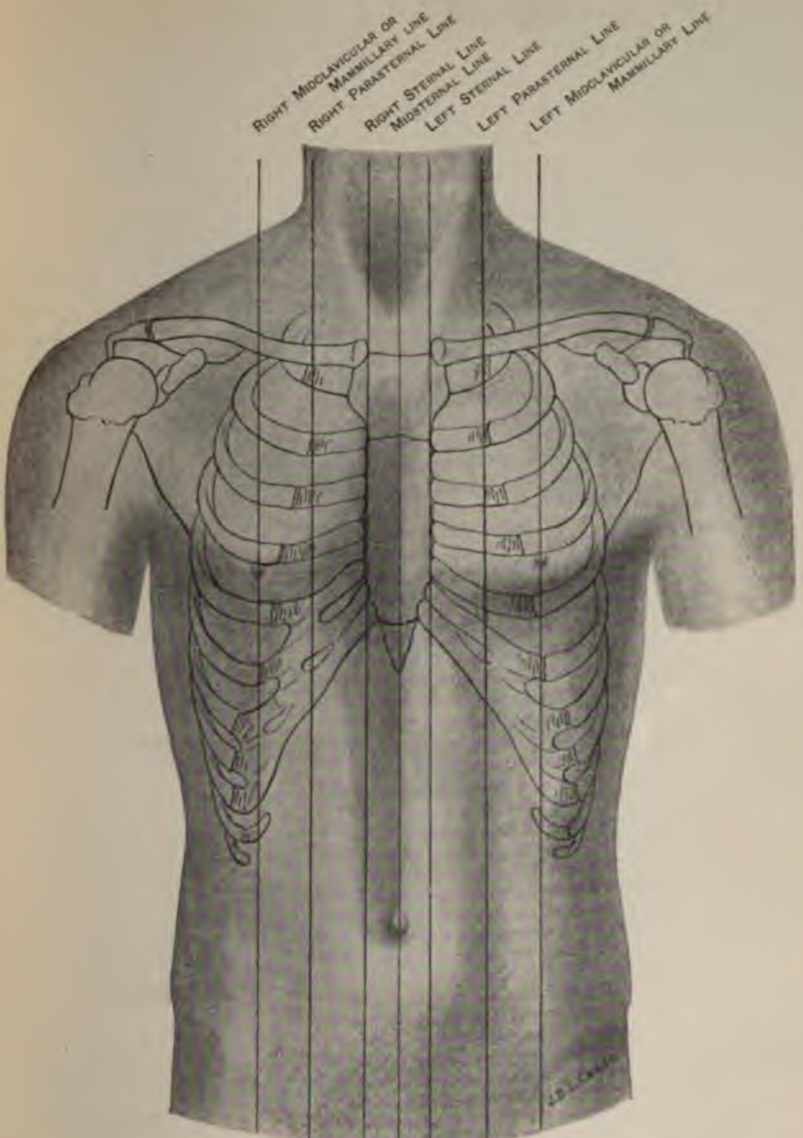
(11) **Suprascapular Regions.**—The boundaries are those of the small, circumscribed, flattened areas immediately above the scapular spines, and limited above by the borders of the trapezius muscles. They correspond to the supraspinous fossæ of the shoulder blades.

Relational Anatomy.—The apices of the lungs are in relation here, which marks these regions of great importance in the detection of incipient tuberculosis.

(12) **Scapular Regions.**—They correspond to the outlines and spaces of the infraspinous fossæ of the scapulæ.

Relational Anatomy.—Lung tissue and the main lobar fissures are opposite these regions.

(13) **Infrascapular Regions.**—They are bounded above by the level of the inferior angles of the scapular bones; below, by the edge of the thorax; posteriorly (inside), by the dorsal spines below the



Bony Landmarks of the Anterior Surface of the Chest, with the Vertical Lines for Measurement and Reference.

seventh vertebra; anterolaterally (outside), by the posterior borders of the infra-axillary regions—at the vertical lines through the points at which the latissimi dorsi muscles leave the chest.

Relational Anatomy.—Just below the surface, on both sides, are the lungs, their borders extending downward as far as the eleventh ribs. On the *right* side, below the lung border, lies the uncovered narrow strip of liver, and, close to the spine, the upper portion of the right kidney. On the *left* side, from the backbone outward, are aorta, kidney, intestine, and spleen.

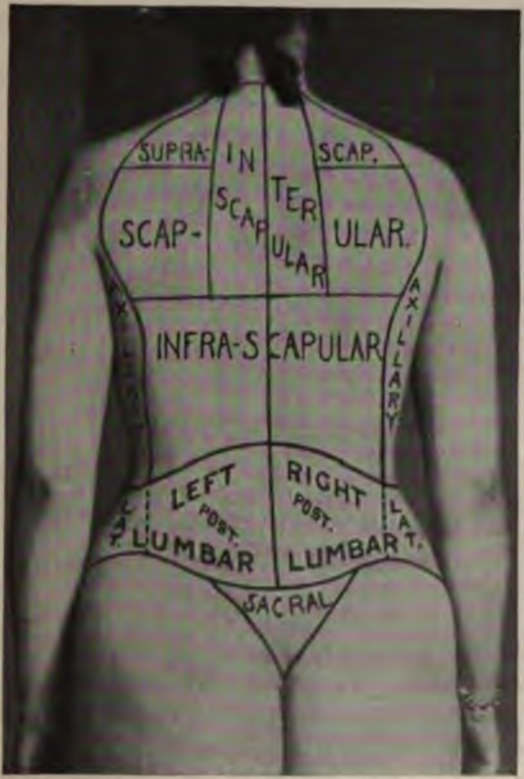


Fig. 2.—Showing the Topographical Areas of the Trunk Posteriorly. (Butler.)

(14) **Interscapular Region.**—This occupies the space between the shoulder blades, and from the second to the seventh dorsal spines—the latter at the level of the inferior angles of the scapulæ.

Relational Anatomy.—On both sides this region contains lung substance, the main bronchi (bifurcation at the fourth dorsal verte-

seventh vertebra; anterolaterally (outside), by the posterior borders of the infra-axillary regions—at the vertical lines through the points at which the latissimi dorsi muscles leave the chest.

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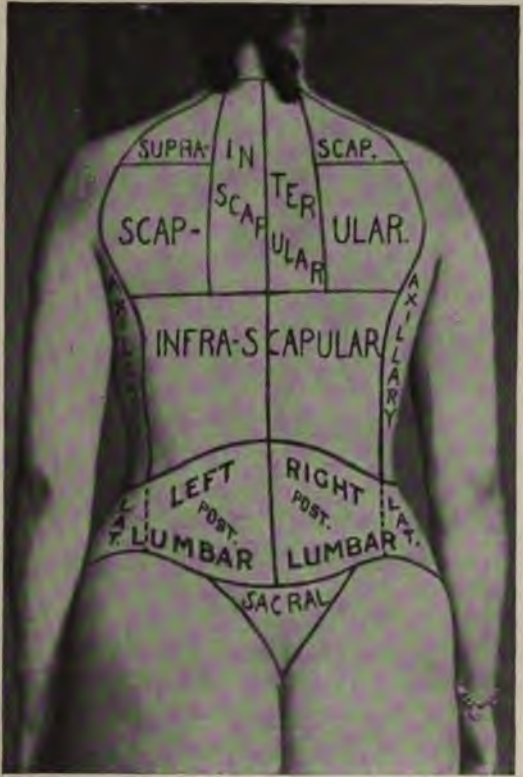


Fig. 2.—Showing the Topographical Areas of the Trunk Posteriorly. (Butler.)

(14) **Interscapular Region.**—This occupies the space between the shoulder blades, and from the second to the seventh dorsal spines—the latter at the level of the inferior angles of the scapulæ.

Relational Anatomy.—On both sides this region contains lung substance, the main bronchi (bifurcation at the fourth dorsal vertebra), and the bronchial glands. On the left side it also contains the descending aorta (from the third or fourth dorsal vertebra), esophagus, and thoracic duct.

RELATIONAL ANATOMY (LANDMARKS) OF THE LUNGS

Before taking up the physical methods of examining the lungs, it is needful to know certain definite facts in connection with the surface relations of the boundaries, limits, fissures, and lobes of the lungs; and also points bearing upon the position and extent of the pleural sacs. There are important differences between the two sides, although on both sides the lungs are everywhere in contact with the thoracic wall, except in the neighborhood of the heart and behind a small portion of the upper part of the sternum, and they both reach from their apices to the sixth ribs in front and to the tenth behind.

Right Lung (see accompanying figures).—UPPER LIMIT: The apex projects into the supraclavicular region from three-fourths to one and three-fourth inches (2 to 5 cm.) above the clavicle, in front; behind, as high as the level of the seventh cervical spinous process.

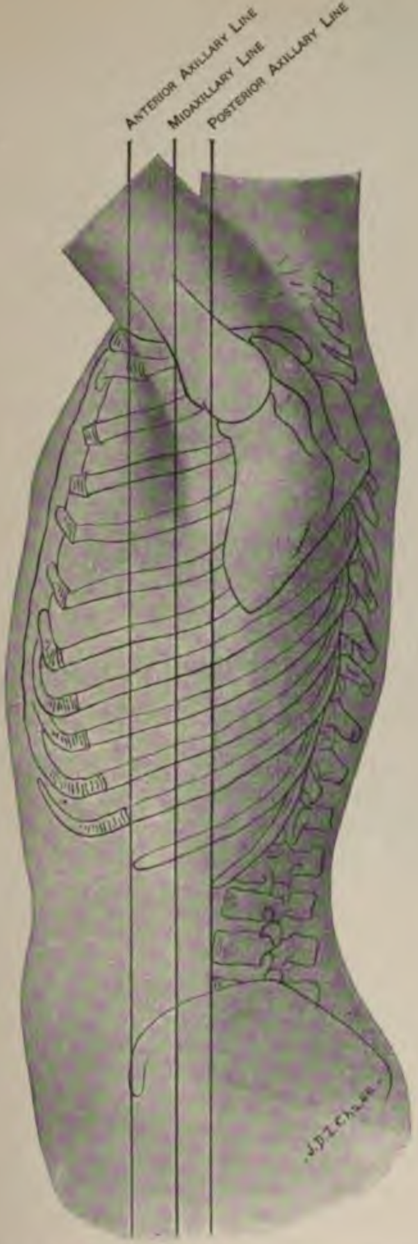
MEDIAN LIMIT: Somewhat to the left of the median line of the sternum, behind the angle of Louis, to which landmark the anterior border converges (downward, forward, and inward) from the apex. It continues thus to the fourth chondral level, then gently outward and downward to the sternal insertion of the sixth costal cartilage, whence it passes more abruptly outward to the

LOWER LIMIT: The now lower border here follows the sixth rib to the mammillary line, then nearly horizontally backward intersects the eighth rib in the middle axillary line, the tenth rib in the scapular line, and reaches the level of the eleventh dorsal spine near the vertebral column.

POSTERIOR LIMIT: This corresponds to the vertebral border, running nearly parallel to the spine from the latter limit to the apex just below the border of the trapezius in front. Practically, it has been customary for me to indicate, as an aid to memory, the even numbers of the ribs passed, as follows: second, fourth, sixth ribs (front), eighth rib (side), tenth rib (back).

Left Lung.—The limits are relatively the same, except that the left apex is apt to rise a trifle higher, and the inferior border to

PLATE VII



Landmarks and Lines of the Lateral Surface of the Chest.

reach a trifle lower, than the right. The only important, and at the same time characteristic, difference is in the notchlike course of the anterior border between the fourth and fifth ribs directly over the right ventricle of the heart. From the sternal end of the fourth cartilage this border curves sharply outward and downward behind the fourth interspace to a little beyond the parasternal line, then slightly inward to the fifth interspace, and again bending abruptly downward and outward to cross under the sixth rib in the mammillary line, as on the right side: the lingula of the lung, so-called, is thus formed.

Fissures of the Lungs.—A knowledge of the fissures—their landmarks—though of secondary importance, is sufficiently needful because of the lobes into which they divide the lungs. This is especially true in cases of lobar pneumonia, where the limitation or progress of the disease to one or more lobes may thus be recognized by the boundary relations of the physical signs.

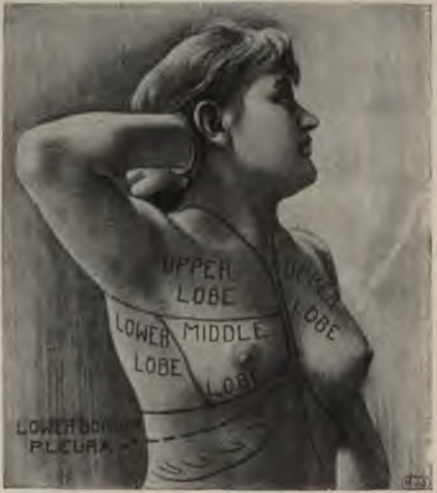


Fig. 3.—Showing the Lobes of the Lung and the Lower Limit of the Pleura on the Right Side of the Chest. (Butler.)

Fissures of the Right Lung.—*The Long or Greater Fissure.*—It separates the middle and upper lobes from the lower. Its starting-point posteriorly is at the vertebral border of the lung at about the level of the inner end of the spine of the scapula (third dorsal vertebra). Its direction is obliquely downward and forward, passing behind the fourth rib in the midaxillary line, and terminating at the antero-inferior edge of the lung at the sixth rib in the mammillary line.

The short or lesser fissure branches off from the preceding at or near the outer border of the scapula and third interspace, and passes nearly horizontally forward, and ends at the anterior edge of the lung under the fourth-rib insertion, thus dividing the upper from the middle lobe.

Fissure of the Left Lung.—This one fissure, separating the upper from the lower lobe, starts, courses in the same direction, and terminates with the same anatomic surface relations as the greater fissure on the right side—i. e., posteriorly, third dorsal vertebra; laterally (axilla), fourth rib; anteriorly, sixth rib.

Lobes of the Lungs.—The position of the lobes is therefore as follows: generally stated, the upper lobes present mainly in front,

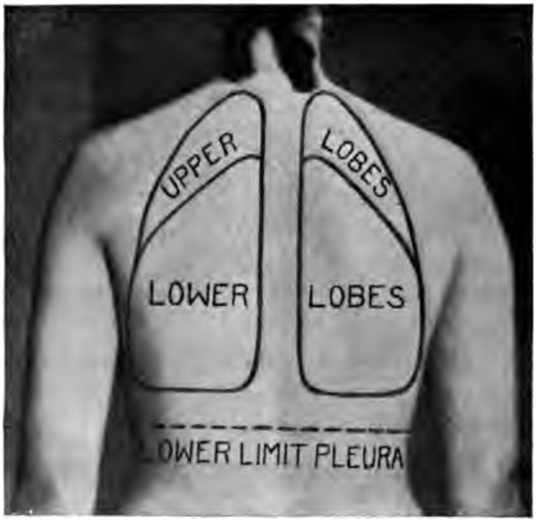


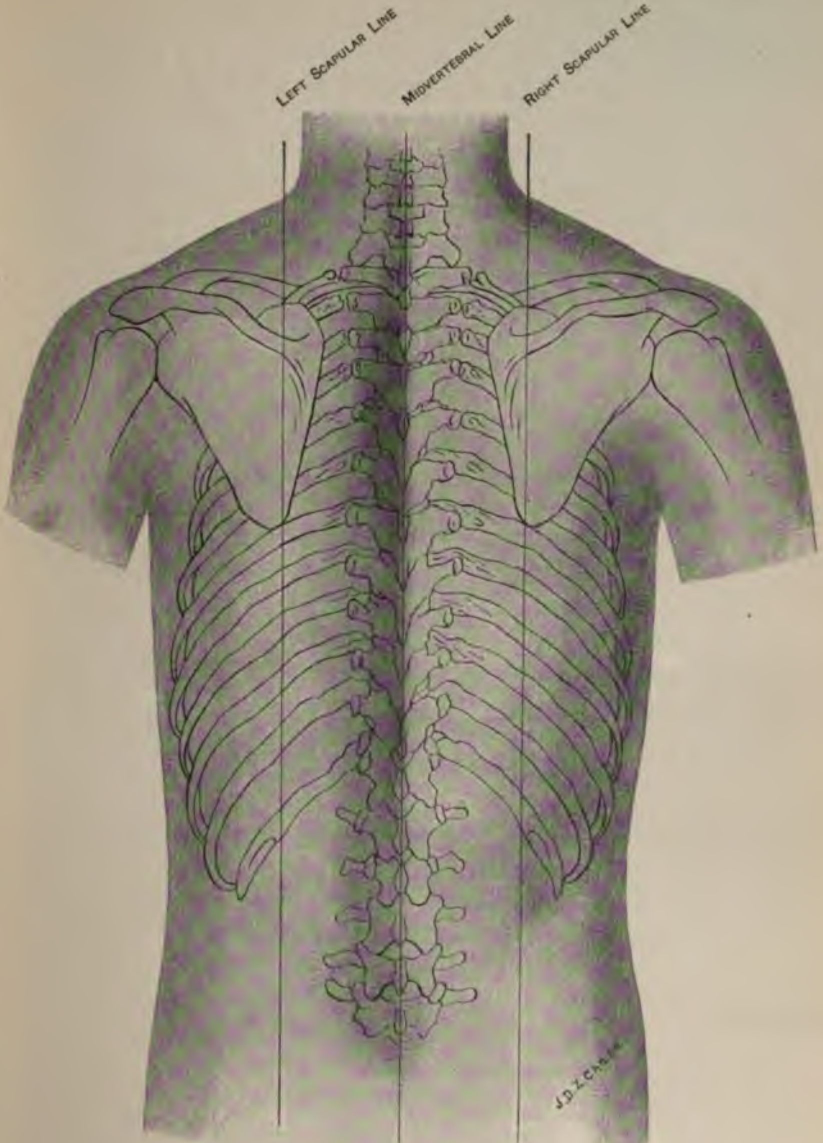
FIG. 4.—SHOWING THE LOBES OF THE LUNGS AND THE LOWER LIMIT OF THE PLEURA POSTERIORLY. (Butler.)

and the lower lobes behind. More specifically: ANTERIORLY, on the right side we have upper lobe as far down as the fourth rib; from there down to the sixth rib, the middle lobe; on the left side, upper lobe practically alone. LATERALLY, at the right, parts of the upper, middle, and lower lobes; on the left side, portion of upper lobe (to fourth rib), and lower lobe downward to eighth rib. POSTERIORLY, on both sides, upper lobe (apices) as far as the scapular spines, below which the rest is lower lobe.

RELATIONAL ANATOMY OF THE PLEURAL SACS (PLEURÆ)

In quiet respiration the borders of the lungs do not coincide with the limits of the pleural sacs, though their boundaries correspond most closely along the anterior edges of the lungs down to the fourth ribs. But along the lower borders of the lungs and at the cardiac

PLATE VIII



Landmarks and Lines of the Posterior Surface of the Chest.

notch on the left side below the fourth rib the pleural spaces extend some distance beyond the former; here these spaces are termed the *complemental pleural sinuses.* With deep inspiration the lungs may expand sufficiently to fill the complemental pleural spaces. Their size depends upon the form and development of the lungs and thorax. The largest space is below the inferior borders of the lungs in the infra-axillary regions, the height being as much as three and one-half inches (10 cm.). In the mammillary and scapular lines the distance is from one and one-half to two inches (4 to 5 cm.).

ADDITIONAL ANATOMIC LANDMARKS

The **bifurcation of the trachea,** it should be remembered, is at the level of the angle of Louis, or second costal cartilage, and of the disk between the third and fourth dorsal vertebræ.

Primary Bronchi.—The *right bronchus* is larger in caliber than the left, shorter in length, more horizontal in direction, and lies under the second rib, whereas the left lies under the second interspace. The proximity of the right bronchus and its main branch to the upper lobe has important bearing upon certain auscultatory phenomena over this region, as will be referred to later.

The surface relations of the lungs to such organs as the heart, liver, spleen, and stomach, which are partly covered by the lungs, will be pointed out as their relations with each other are developed according to the pathologic conditions affecting them.

Supplemental to the preceding, it should be noted that the summit of the **diaphragm** is as high as the level of the fourth rib on the right side (about an interspace higher than on the left), the inferior surface of the lung capping it, and extending outward and downward to a wedge-shaped edge at the sixth rib in the mammillary line, etc.

CHAPTER II

INSPECTION

Definition.—Inspection is the act of looking intelligently and attentively, and is naturally the first method in examining a patient's chest, and by it the physical evidences of thoracic disease are obtained with more or less clearness, directness, and positiveness. In fact, the student should avoid neglecting or slighting this old and simple method because of the attractiveness of the more modern, but more difficult and involved, if precise and penetrating, methods of percussion and auscultation. Undue haste and self-confidence on the part of the examiner, and false modesty or a stubborn fastidiousness on the part of the patient may, of course, tend to develop the pernicious habit of lack of thoroughness in omitting a careful and complete ocular examination.

Method of Procedure.—No chest can be inspected unless it be exposed. As most of the regions of the thorax, as well as the appearances of the thorax in its entirety, must be noted in any proper examination of the thoracic organs, it is usually necessary to have the patient stripped to the waist, whether standing, sitting, or lying. Of course, circumstances may frequently arise to modify this principle. Thus, in examining females a sense of propriety, or with the very delicate and sensitive physically, or the very ill, a motive of precaution will suggest the exposure of one aspect of the chest, or its greater portion, at one time. Again, after the seat of disease has been detected by a thorough examination, it is often sufficient at subsequent meetings to limit the examinations to this locality.

A good, steady, white light (preferably daylight) is of first importance. The patient should lie, stand, or sit on a perfectly even plane; the position should be quite comfortable, relaxed and easy, the arms allowed to hang loosely by the side. The sitting or standing postures should be selected whenever possible. As a rule, the light should fall directly and symmetrically upon the surface to be examined, the examiner standing with back to the light. A side or slanting light falling obliquely upon the surface examined by turning the patient or moving the source of illumination is often advisable, however; abnormal bulgings and depressions, and the moving shadows of abnormal pulsations, are thus readily detected, whose diagnostic importance may be considerable. Inspection should be practised anteriorly, posteriorly, laterally (the arms being raised), and superiorly; the latter, by standing behind or alongside the patient, enables the physician to estimate approximately the anteroposterior diameter of the chest, its outline on an imaginary horizontal section, and further accentuate any general or circumscribed abnormalities of size, contour, or movement. The patient's body and members should be quiet, the breathing easy and uninterrupted by conversation. Always, observation should be comparative; that is, both sides habitually compared generally and in respect to corresponding regions. In the comparison, allowances must be made for the obviously natural differences, as of greater muscular development on one side, and for certain physical and accidental irregularities that may be peculiar to each individual examined.

We begin with the visible characteristics of structure and movement of the normal thorax, in order that the physical evidences of thoracic disease may more surely be discriminated.

THE NORMAL THORAX

The ability to recognize the normal thorax distinctly and readily depends upon one's general observation and clinical experience; in familiarity with the proportions, forms, and movements of the bared chests of athletes, swimmers, laborers, and others of manifestly average health and physical development, including patients examined while consulting for ailments other than thoracic, or for slight or obscure chest troubles without external physical alterations.

Perfect symmetry of chest is rarely found. The regularly constructed and well-nourished chest of the adult male, viewed anteriorly, and exclusive of its shoulder girdle, is conoidal in shape, having the smaller end uppermost; with the shoulder attachments, however, the chest appears larger at the level of the axilla than at the ensiform cartilage; the two sides are both generally and regionally symmetrical; the clavicles are slightly prominent, and the supra- and infraclavicular depressions slightly noticeable; there are small depressions, one above the suprasternal notch between the inner ends of the clavicles and one (infrasternal) over the xiphoid process; the intercostal spaces are visible only at the lower and lateral ribs; the sternum in profile is nearly straight; the angle of Louis (formed by the union of the manubrium and gladiolus of the sternum) is slightly but distinctly marked at the landmark level of the junction of the second rib; the sternovertebral (anteroposterior) diameter is obviously a little shorter than the transverse (about one-fourth less: see Mensuration), the shape of a transverse section being elliptoid; the ribs so leave the sternum that from above downward their course changes gradually from the horizontal to a decided obliquity, the coalesced costal cartilages of both sides forming at the xiphoid nearly a right angle (*epigastric or subcostal angle*); posteriorly, the scapulæ lie flat upon the thorax, in the upright position, and are so highly placed that the shoulders stand out nearly horizontal from the neck; the spine is straight or but very slightly curved to the right at mid-back, and the vertebral sulcus more or less deep, according to the fatness or thinness of the individual.

Although this ideal form of thorax is seldom met with, the deviations about to be mentioned commonly occur in persons of sound general health and thoracic organs. These compatible irregularities, or "physiological heteromorphisms"—departures (M. Woillez)—are principally as follows: (*congenital*) a form of thorax with shallow upper but gradually wider and deeper lower zone; a shorter thorax with, at the same time, acute epigastric angle; marked prominence of the clavicles, with deepened supraclavicular spaces on both sides (relative); marked angle of Louis; greater prominence of one or several ribs, especially the second, third, and fourth, in front, with increased curvature, or the lower ribs may be pressed in and flattened; finally, we sometimes find the ensiform cartilage projecting decidedly forward or backward; (*acquired*) the right side may be larger than the left, owing to greater muscular development, as in brakemen, blacksmiths, and carpenters; one shoulder may be a little lower than the other, and the dorsal spine curved slightly, usually to the right, as in journalists, litterateurs, clerks, tailors, hod-carriers, and the like. Again, there may be slight differences in the relations between the natural diameters of the chest. Partial or local defects of symmetry are also discovered due to previous fractures and dislocations of clavicles and ribs, and all may be compatible with perfectly normal lungs and heart.

Normal Chest Movements.—Whether purposely forced, or involuntary and quiet, the normal breathing movements are regular, rhythmical, gradual, and symmetrical—quite equal and even on both sides. Observation of these general thoracic movements has direct reference to the inspiratory and expiratory functions of the lungs, the former characterized by a simultaneous expansion and elevation of the thorax and the latter by a simultaneous retraction and depression; the former active—that is, produced by diaphragmatic and intercostal muscular action principally; the latter passive, the result of the elasticity of the lungs, the weight of the chest wall, and the resilient pressure of the abdominal organs against the diaphragm.

By inspection we learn, then, the *size*, *shape*, and *symmetry* of the chest; the *rate* or *frequency of respiration* (more often an objective symptom), and the *rhythm*, *degree*, and *character* of the *breathing movements* as physical signs.

Types of Normal Respiratory Movement.—Before describing the preceding features, it should be borne in mind that there are in health two types of respiration, named according to the predominance of either one of the two larger elements seen in all normal respiratory action. These are (1) the *superior thoracic* or *costal* type and (2) the *abdominal*, *diaphragmatic*, or *inferior costal* type. Abdominal respiration is more marked in men and in children of both sexes; superior costal respiration in women. In the costal type of breathing, the upper anterior part of the thorax swells out more fully with each inspiratory enlargement; the lower costal and ventral movements are comparatively limited. The *costo-abdominal* or diaphragmatic type of breathing—the ordinary, calm breathing of adult males—predominating, we notice the characteristic swelling projection of the abdomen, especially the epigastrium, simultaneous with the general expansion of the chest, and thus produced by the contraction and flattening of the diaphragm pushing the intestines against the belly wall. The preponderance of either type of breathing is modified somewhat by the following influences: *Age*, determining the degree of flexibility of the bony case, permits in early youth, for example, greater relative freedom of pectoral movement in the male, also more general action and less limitation to the upper regions of the thorax in the female; however, the marked evidences of the superior costal type of respiration, even in very young girls, show (as do those of the abdominal type in boys) the fundamental sexual differences of *heredity*. Again, in aged women, with firm and more or less unyielding thoracic walls, the abdominal movements are increased. During *sleep* the costal breathing is more pronounced in men as well as in women; this observation is true also when voluntary *forced breathing* is practised, as in singing; indeed, there may be habitual exaggeration to a sligh degree of the upper costal action in professional singers even durin quiet breathing. There is no doubt that the custom of wearing tightl laced and rigid *corsets* is partly responsible for the excess of uppe chest movement seen in most women.

In regard to the thoracic breathing, it should be noted that th diverging movement of expansion, with increase of the horizonta diameters, is visibly more decided than that of elevation of the ribs but that the energy and extent of both are directly proportionat to each other under all circumstances of health. The movements o the *intercostal spaces* are distinguishable according to the thinnes of the individual, the regions observed, and the force of the breathing In the male, the interspaces are most visible in the infra-axillar regions; in the female, in the infraclavicular regions. They ar slightly hollow both during inspiration and expiration, though mor so during the former, and, naturally, markedly so in forced breathing

Finally, the respiratory movements may be altered in the norma chest by *posture*. Thus, when lying on one side, in calm breathing the motion of that side is interfered with, resulting in compensator exaggerated movement on the opposite side. The fact that eithe the costal or diaphragmatic type of respiration may be exaggerate in certain diseases makes it important to recognize these types an their conditional variations in health in order that the pathologica may be estimated better.

THE DIAPHRAGMATIC PHENOMENON (Litten's Sign).—Inspectio of this moving indicator of the action of the diaphragm require special consideration becaus of its diagnostic value. Th sign itself, as well as th method of observing it, wil be understood by a brief an diagrammatic reference t the anatomy and physiolog of the diaphragm. The accompanying diagram (fron Cabot) will aid in understanding the mechanism o the production of this phenomenon—a moving shadow. As at the end of expiration the diaphragr lies in close apposition to the walls of the thorax from the sixth rib dow to its attachment, during inspiration it descends, and, separating fron

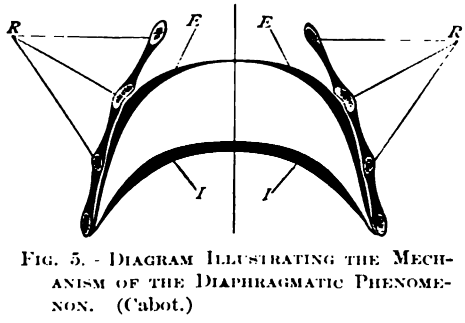


FIG. 5. - DIAGRAM ILLUSTRATING THE MECHANISM OF THE DIAPHRAGMATIC PHENOMENON. (Cabot.)

the ribs, leaves room for the lung to descend also. This " peeling off " of the diaphragm causes a shadowy, wavelike retraction of the intercostal spaces of the lower lateral aspects of the chest, due probably to the external pressure exceeding the intrapulmonary at the beginning of inspiration, the balance of pressure being restored as soon as expansion of the lung is completed. Although present in all healthy individuals, it is best seen in those who are thin and muscular. The phenomenon is best observed in the following manner: the patient lies flat on his back, with bared chest, and face looking directly toward a window or strong night light, the head being slightly elevated; side lights should be carefully excluded. The observer stands at a distance of a few feet from the patient's side, with back to the light, and views at an angle while the patient takes a full, deep breath. With the beginning of inspiration a small, narrow, wavy shadow is seen to move down the axilla (on either side) from the sixth rib or seventh interspace to the ninth or tenth ribs, or even to the borders of the ribs. The movement of the shadow in normal chests is about two and one-half inches; with strongest breathing, about one inch more. The rising movement of the shadow during expiration is less distinctly visible than the falling, inspiratory one. I have observed this phenomenon in the epigastrium in several instances. As will be pointed out later, the absence of this sign is suggestive of certain affections of the lungs and pleuræ that interfere with the action of the diaphragm.

It may also serve as an index of a person's respiratory capacity, according to the measured excursion of movement as compared with the general chest effort; thus, as intimated by Cabot, the use of the spirometer, as well as of the X-rays, in observing the diaphragmatic movements may be compensated for whenever their impracticability or expensiveness are obstacles, or, as often happens, when they are practically unnecessary.

Frequency of Respiratory Movements.—Though not strictly a physical sign in itself, the rapidity of breathing is so closely related to pathologic conditions of the thorax of marked physical characteristics that the normal rate and its physiologic variations must first be noted. In healthy adult males the respiration rate is from 16 to 22 per minute; in females it may be from 18 to 24; in children of five years, about 26; in the new-born and under one year, about 44. Besides age, the rapidity of the respirations may be influenced by other intrinsic and certain external conditions: it is greater in walking, standing, and sitting than in lying; it is increased after meals, and with bodily exercise, mental activity, and emotional excitement. It is likewise more marked in spring than in midsummer, except when excessive heat acts upon the temperature of the body; during the day than at night, and in the rarefied atmosphere of elevated regions than in the lowlands. As the frequency of the respirations may be altered subconsciously in persons who are temperamentally sensitive while under observation, it is my custom to count the breathings before relinquishing attention to the pulse-rate or to the timing of the thermometer. Of course, during sleep a more correct estimate may be obtained in many cases. The epigastric as well as the thoracic rise and fall may be watched in counting the respirations, and aided often by applying the hand or by noting the breath sounds.

NORMAL RESPIRATORY RHYTHM.—The relative duration of inspiration to expiration in quiet breathing is visibly about equal; physiologically and more precisely, however, the ratio is about as 6 to 7. Inspiration begins suddenly and advances rapidly, then slackens; expiration follows so closely that no distinct pause is perceptible between it and inspiration; expiration begins rapidly, and slows gradually to the close. The pause at the end of expiration is more observable the slower the respiration. The regularity of the successive respirations may be varied normally in forced and hurried breathing, in which the inspiration becomes relatively shorter in duration. In children the rhythm is also very variable, whether asleep or awake. Pathologic alterations of rhythm are usually associated with the physical evidences of labored breathing, or dyspnea.

The DEGREE OF NORMAL RESPIRATORY EXPANSION is more precisely determined by mensuration than by either inspection or palpation. It is affected in general by the degree of pulmonary development and exercise, and must be equal on both sides.

Nutrition.—The chest in health shows a sufficient amount of flesh and subcutaneous fat so that the clavicles, ribs, and scapulæ are nicely covered and not too prominent; nor, on the other hand, that the shallow depressions do not sink in too much. Emaciation is closely related to various affections of the lungs and pleuræ.

MENSURATION

(*Thoracometry, Cyrtometry, Spirometry,* etc.)

It seems to me a mistake to subordinate the relative importance of thoracic measurements, as is so commonly done. With the growing adoption of diagnostic methods of precision, this most precise one—*mensuration*—deserves at once increasing scientific study and practical application. As a method of physical examination it has special value to those physicians—and they are becoming numerous—who perform the responsible and exacting duties of medical examiners for life-insurance companies, pension boards, railroad corporations, and the like, or who serve as physical directors to the various educational and philanthropic institutions (Y. M. C. A.'s) conducting well-equipped and modernly-managed gymnasiums.

As the results obtained by inspection are so intimately related to—being confirmed or modified by—those derived by mensuration, the latter requires consideration at this point.

The object of measuring the chest is to ascertain more accurately than can be done by inspection or palpation the general size or bulk and shape, the comparative size and shape or symmetry of the two sides, the relative positions of different parts and their distances from fixed points, with the thorax at rest, and to determine the degree and variations in the bilateral, unilateral, or local expansion and retraction accompanying the movements of inspiration and expiration.

Ordinarily, the circumference and semicircumference of the chest are measured with the tape, and the principal diameters with the thoracometer or calipers (such as obstetricians use for pelvic measurements).

Thoracometry.—The *circumference* is the main measurement, taken usually at the level of the nipples in men, or a little above, at the third costosternal articulation, just at the upper edge of the mammæ, especially important in women. Care should be taken that corsets and all chest coverings except a light, loose undergarment are removed, the body in a resting, easy, upright position, and that the tape is horizontally and evenly applied around the thorax. In this way the general size and degree of expansion of the chest are ascertained.

The average circumference in men (repose) is about 34.3 in.; in women, 30 in. There are many variations, of course, in healthy individuals from these approximate measurements, depending upon age, height, weight, or the relation to the development of the rest of the body. The extremes are usually between 27 and 44 in.

Table showing Size of Chest in Relation to Height and Weight.

Height.	Chest.	Weight (Standard).	Twenty per cent under weight.	Forty-five per cent over weight.
5 ft.	33 in.	115 lbs.	92 lbs.	167 lbs.
5 " 1 in.	34 "	120 "	96 "	174 "
5 " 2 "	35 "	125 "	100 "	181½ "
5 " 3 "	36 "	130 "	104 "	188½ "
5 " 4 "	36½ "	135 "	108 "	195 "
5 " 5 "	37 "	140 "	112 "	203 "
5 " 6 "	37½ "	143 "	114 "	207 "
5 " 7 "	38 "	145 "	116 "	210 "
5 " 8 "	38½ "	148 "	119⅛ "	215 "
5 " 9 "	39 "	155 "	124 "	224½ "
5 " 10 "	39½ "	160 "	128 "	232 "
5 " 11 "	40½ "	165 "	132 "	239 "
6 "	41 "	170 "	136 "	246 "
6 " 1 "	41½ "	175 "	140 "	253 "
6 " 2 "	42½ "	180 "	144 "	260 "

Respiratory Expansion.—This important measurement is also taken with the tape, and represents the difference between the circumference at the end of a forced inspiration and of a forced expiration. During calm respiration the circumferential expansion is very slight—hardly more than one-half of an inch in a well-developed male adult. The extremes of expansion vary normally from 2 to 5 in.; the average in men is about 3 in.; in women, 2½ in. An applicant for life-insurance with an expansion of less than 2 in. is likely to be rejected as an unfavorable risk. It should be borne in mind that many persons with healthy chests are often unable to produce their fullest expansion owing to embarrassment or lack of coordinating power; on the other hand, some less robust persons who are practised in deep breathing or in accessory muscular action may easily expand 4 or 4½ in., while certain phenomenal athletes may develop as much as 7 in. of expansion. The amount of expansion is influenced by posture, being least in recumbency, more in sitting, most of all in the erect, standing position. Forced breathing has no more effect upon the abdominal expansion than is mensurable around the thorax during ordinary quiet respiration—that is, about ¼ to ½ in.

Semicircumference.—This may be obtained accurately for both sides by the use of two tapes joined at the commencement of their scales, and padded slightly a little beyond the point of junction so as to rest like a saddle piece over the spine. The measurements are taken simply by passing each tape around its respective half of the chest to the midsternal line and reading off the separate inches or centimeters, as may be marked. The right half measures normally (at the nipples) from ¼ to ½ in. more than the left in right-handed persons; in left-handed persons the left semicircumference may equal or slightly exceed the right (¼ in.).

The UNILATERAL EXPANSION of the chest may be measured also by noting the differences on the scales for each side with complete expiration and inspiration.

The DIAMETERS of the chest may be measured by means of the thoracometer or caliper compasses. The *anteroposterior* or *sterno-vertebral* diameter is taken by applying one blade of the calipers on a level with the nipple, in the midsternal line, and the other at the insertion of the second rib behind. The *transverse* diameter is measured at the highest point of the axillæ, and in the normal chest is a little greater than the antero-posterior. The average depth of the chest is about 7½ in. in men, about 7 in. in women; the breadth averages about 10 in. in men.

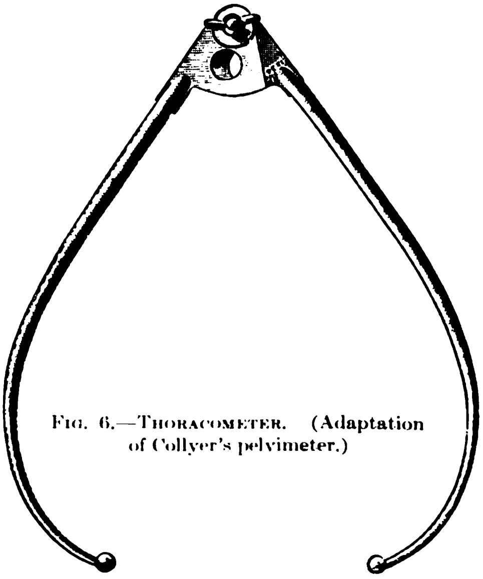


FIG. 6.—THORACOMETER. (Adaptation of Collyer's pelvimeter.)

The LENGTH of the thorax may be ascertained by measuring from the clavicle to the lowest border of the ribs, along the midclavicular line. As a basis for estimating chest length and variations therefrom, the *linea costo-articularis* (line drawn from the sternoclavicular articulation to the tip of the eleventh rib) may be used also.

Cyrtometry.—This determines the shape of the chest by several means of outlining, usually on a level with the nipples, though it may be desirable sometimes to represent a transverse section of the chest at any higher or lower plane.

The cyrtometer of Woillez, often referred to by authors, is of historical rather than practical interest. It consists of a chain of stiffly moving links, which may be closely applied to the circumference of the chest and carefully removed so as to preserve its outline when placed on a table, and its characteristics and diameters noted. A simple and sufficient instrument for ordinary clinical purposes consists of two narrow, tapelike, leaden strips joined by a stout leather hinge, or with very short flexible attachments, to a padded metal vertebral piece. The latter being held firmly over the spine at the desired level, the strips are molded accurately around the chest and pencil-marked where they cross anteriorly, in the middle line. After carefully removing the pieces so as to hold their shape, the contour may be delineated on a sheet of paper by laying them thereon, being sure that the sternal ends are crossed as marked; the outline may then be traced inside the borders of the strips. The form of the cross-section of the chest may be traced around the diameters obtained previously with the calipers and marked on the paper, thus completing the mensuration.

CHEST PANTOGRAPH.—This new instrument for recording chest contours was contrived by Dr. W. S. Hall, of Northwestern University, Illinois. It is a modification of the pantograph used by artists for the purpose of enlarging figures from smaller copies, whereas this reverses the process. The instrument is made of wood or brass, with a brass or steel semicircle.

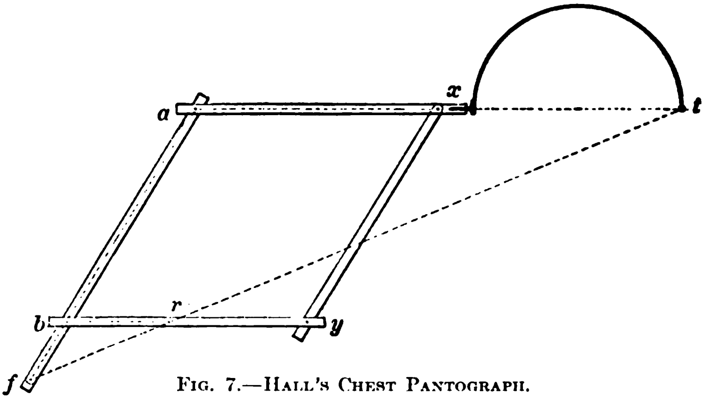


FIG. 7.—HALL'S CHEST PANTOGRAPH.

Hall describes the method of working as follows: "The joints *a*, *b*, *x*, and *y* move easily in the plane of the instrument. The semicircle, 40 in. in diameter, rotates at *x* around the diameter *tx*. The point *f* is fixed to a table. With *f* a fixed point, all movements of *t*, the tracing point, are accompanied by corresponding movements of *r*, the recording point. The triangles *f*, *r*, *b* and *f*, *t*, *a* are similar triangles in all positions of the instrument; *fb*:*fa*::*fr*:*ft*; but *fb*:*fa*::1:5; therefore, the distance *fr* is always one-fifth the distance *ft*.

"The object of the semicircular arm is, of course, to permit the tracing point (*t*) to be carried around the thorax. The seat upon which the subject sits is adjustable in height, and has back and side supports for the waist, so that the upper part of the body is not allowed to waver from side to side, distorting the contour. If the subject to be examined sit beside the table on which the instrument is fixed, if the seat be adjusted in height to bring the plane of the thorax to be examined into the plane of the instrument—i. e., on a level with the top of the table; if a sheet of millimeter paper be fixed to the table under the recording pencil *r*, and if the tracing point *t* be swept around the thoracic wall, a record of the chest contour will be traced upon the paper."

Three pantograms represent outlines taken at the axillary, nipple, and abdominal planes respectively.

Additional points of value that may be studied are the relative increase in the anteroposterior and lateral diameters in forced inspiration, the differences between the cross-sectional areas (in centimeter squares) of expiration and inspiration, and local prominences of musculature, bony irregularity, or pathological altera-

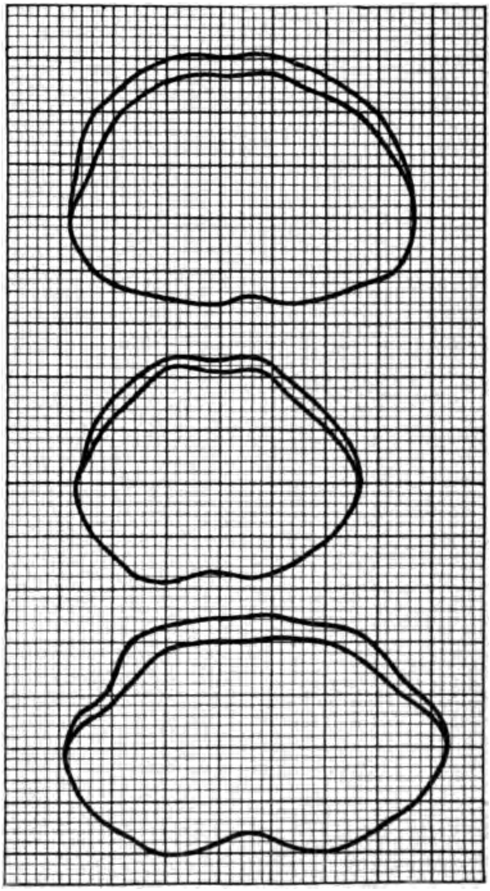


FIG. 8.—CHEST PANTOGRAMS, TAKEN AT THREE PLANES. (W. S. Hall.)

tion of contour. These graphic records of chest contour, reduced in size, are well adapted for filing with the general notes of a case, and pantograms taken at successive intervals lend themselves readily to a more accurate knowledge of thoracic changes.

It has been demonstrated quite recently by Malone (*Journal Am. Med. Assn.*, September 17, 1904), in a diametric, pantographic, and mathematic study of a large number of chests, that broad chests have a greater cross-sectional area, both for inspiration and expiration, than deep chests; also a greater area expansion and a greater capacity (volume expansion) than do deep chests.

Spirometry.—The employment of Hutchinson's spirometer gives us an idea of the absolute amount of air which circulates in the lungs between the extremes of deepest respiration—the ***total breathing volume***; while circular mensuration of respiratory movements indicate only relative amounts in different individuals. Thus, with this instrument, which is constructed on the principle of a gasometer, we ascertain one's *vital capacity*—i. e., the quantity of air which can be expelled with the deepest expiration after the deepest possible inspiration. The vital capacity is diminished in all pulmonary diseases, although there are no diagnostic or characteristic differences in the vital capacity among these various diseases.

The physical conditions which influence the vital capacity are very numerous and complex, even in health. Perhaps weight and stature bear the most constant relation to the vital capacity, especially stature, although there may be a wide interval between extremes in healthy persons. According to Otis, the average lung capacity for each inch of height is 3.52 cu. in. For a height of 67 to 68 in. inclusive, the lung capacity is about 237 cu. in.; for a height of 69 to 70 in., about 259 cu. in.; for women of about nineteen or twenty years of age, with a height of 62 to 63 in., the capacity of the lungs averages from 145 to 150 cu. in. In children and in the aged it diminishes.

Von Ziemssen and Klemperer assert that the normal ratio is about 22 c.c. for each centimeter of bodily length; and that if the ratio of height to capacity was less than 1 to 20, or, in women, less than 1 to 17, there was probably marked respiratory disturbance.

COMPLEMENTARY AIR, that which may be inhaled by a forced, after an ordinary quiet, inspiration, averages about 1,500 c.c.

RESERVE or SUPPLEMENTAL AIR, or that which may be exhaled by the deepest, after ordinary, expiration, also equals about 1,500 c.c.

Tidal or breathing air, that which is changed by each calm respiration, amounts to about 500 c.c.

Pneumatometry determines the inspiratory and expiratory pressure of air. The pneumatometer as devised by Waldenburg is a modified mercurial manometer. It is found in health that the expiratory pressure is always greater than the inspiratory, exceeding it by about 20 to 30 mm. The results are more variable than those obtained by spirometry.

Stethography, or the instrumental tracing of the movements of the chest walls (Ransome), and **pneumography**, also the recording of such movements (Marey), are of much greater service in physiological than in clinical research.

THE PATHOLOGIC THORAX

In the inspection and mensuration of the thorax as a whole, and independent of its movements, the observation of three features requires first consideration—viz., *size*, *shape*, and *symmetry*. A methodical habit of noting these may readily be acquired by remembering the alliterative *s*.

(1) SIZE

As indicated previously, the visible and mensurable dimensions of the chest vary considerably in different persons enjoying good health. Pathologic disproportions in size may be *general*, *unilateral*, or *local*. As one-sided and circumscribed deviations are obviously asymmetries, they are described as such under the third head. Of course, abnormal sizes of chest are always more precisely estimated by mensuration than by inspection alone.

Pathologic Enlargement.—Abnormal increase in the size of the thorax as a whole may indicate predisposition to pulmonary disease, or an incipient as well as fully developed gross morbid condition. This is frequently observed in those whose occupation necessitates a certain amount of habitual strain upon the air vesicles, as in glass-blowers, wind-instrument players, etc. Mountaineers and others living constantly in very high altitudes, and asthmatics, may show slight or moderate enlargements even when the other physical signs of true emphysema may be wanting. The latter condition (permanent dilation of the vesicles of the lungs) is, however, the chief affection in cases of marked increase in all of the diameters, especially when most prominently involving the upper half of the chest. Both vertical measurements are at the same time usually shorter than normal.

Pathologic Smallness.—This is particularly noticeable in persons predisposed to or actually having tuberculosis; in those who, in childhood, were considered " delicate " or " scrofulous," or mouth-breathers because of adenoid growths in the nasopharyngeal vault, or, as infants, were victims of marasmus, rickets, and other wasting diseases. Small chests are also seen in patients who have suffered long from prostrating and more or less malignant illnesses.

(2) SHAPE

We learn the shape of the thorax by intelligent observation in general, by practise in the estimation of the relative lengths of the anteroposterior and transverse diameters, and more precisely by measurements with the thoracometer and tracings with the cyrtometer and pantograph.

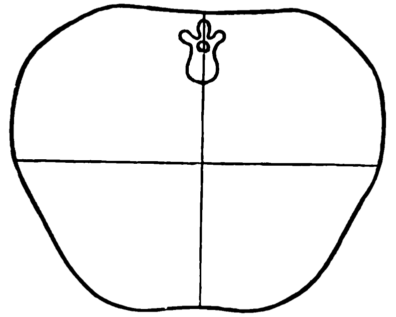


FIG. 9.—THE NORMAL THORAX; OUTLINE OF TRANSVERSE SECTION.

There are certain ***bilateral*** or ***symmetrical abnormalities*** of shape so well marked as to constitute distinct types of pathologic chest. They may originate from congenital, developmental, or previous pathologic conditions.

Paralytic or Phthisinoid Chest.—It is abnormally long, narrow, and shallow; the shoulders slope and droop downward and forward; the neck is long and the larynx and angle of Louis prominent; the ribs are slender, and slanting sharply downward from the sternum, then also with marked obliquity backward to the vertebræ form an acute epigastric angle; the supra- and infraclavicular fossæ and the intercostal spaces are quite depressed, and the latter often widened. Owing to the weakness of the shoulder-girdle muscles, the serratus anticus in particular (" paralytic " thorax), the vertebral borders of the scapulæ project so that the interscapular region may be relatively deepened to the thickness of an applied hand, the scapulæ thus standing out winglike, and hence the designation *alar* or *pterygoid* chest sometimes used.

This type of thorax is seen in delicate children and adolescents, predisposed to or in the incipiency of tuberculosis, although it is not rarely found in those whose lungs have escaped infection. It indicates congenitally small and weak pulmonary capacity. The supervention of tuberculosis causes increase in the flattening of the sterno-vertebral diameter.

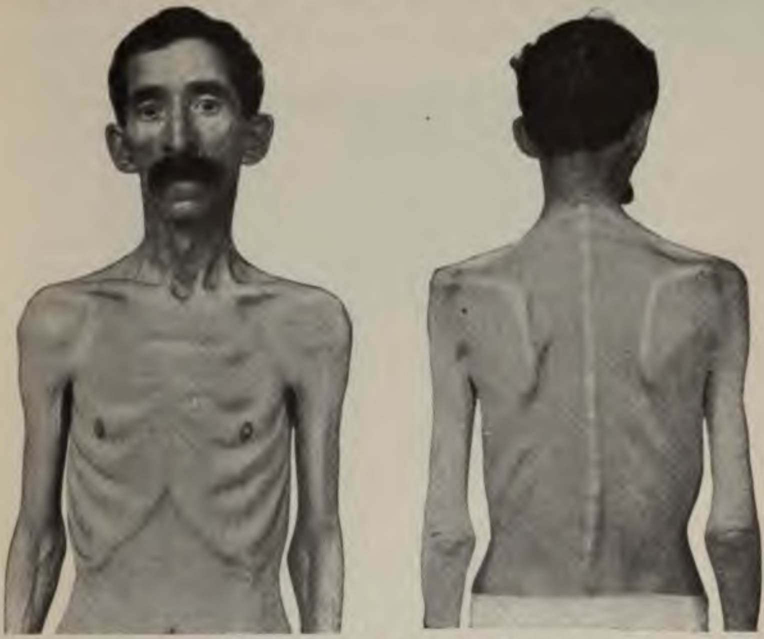


FIG. 10.—ANTERIOR AND POSTERIOR ASPECTS OF THE PHTHISINOID CHEST. (Sahli.)

Flat Phthisical Chest.—The physical characteristics are not much unlike those of an emaciated chest of normal proportions except that the upper anterior aspect shows decided flattening and consequent relative shortening of the anteroposterior diameter. It points to acquired tuberculosis in spite of the absence of the inherited susceptibility usually associated with the preceding type of chest. Furthermore, as Woods Hutchinson has mentioned, pulmonary phthisis may be met with in chests with

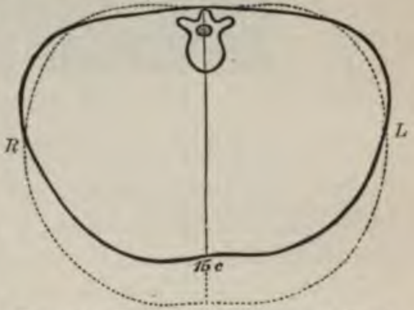


FIG. 11.—THE FLAT PHTHISICAL CHEST. (Gee.)

relatively normal or even slightly exaggerated anteroposterior diameters.

Emphysematous or Inflated Chest.—This is quite the opposite of the paralytic or flat chest. It is a permanent bilateral enlargement of the thorax, and its appearance may be simulated in the healthy chest while at the height of a full inspiration. We see the abnormal fulness and roundness, mainly above the level of the ensiform cartilage, the anterior surface prominent, the dorsal spine bent backward, as seen best from the patient's side, and the marked increase of the anteroposterior diameter, equaling or exceeding the transverse. The measurements and cyrtometric or pantographic outlines confirm inspection, resembling the more circular shape of the normal chest of a child. The neck is relatively short, due to the raised shoulders and thoracic case; the sternomastoids and

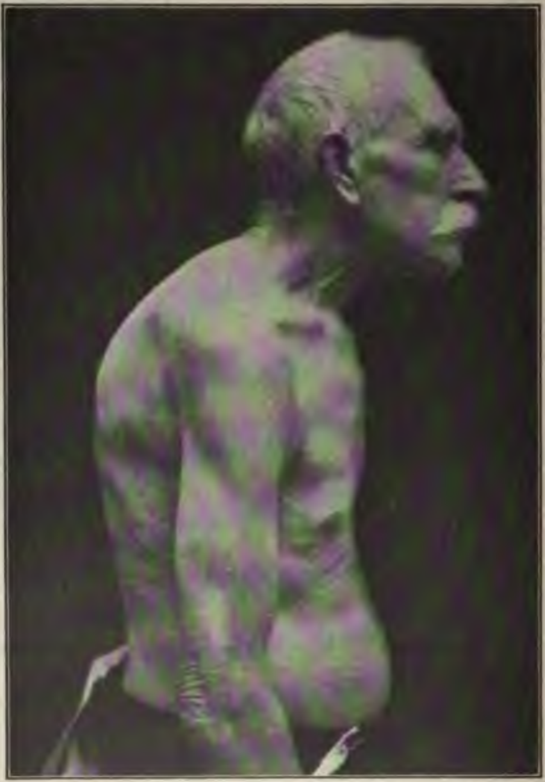


FIG. 12.—EMPHYSEMATOUS CHEST.

scaleni antici muscles may be visibly tense, and the chest as a whole is also diminished in length. The sternum is arched forward; the ribs are thick, hypertrophic, massive looking, and run horizontally outward, consequently the epigastric angle is wider—more obtuse—than normal; the intercostal spaces above are often full, while the lower zone of the thorax is frequently retracted, especially during inspiration. The supraclavicular regions are sometimes deepened and sometimes cushiony. In many cases the lower lateral and infrascapular regions bulge outward and backward, giving rise to the so-called "barrel-shaped" chest. However, the absence of this very characteristic type of thorax does not prove the absence of emphysema in all cases. It indicates permanent overdistention of the vesicles of the lung, caused by prolonged or oft-repeated attacks of chronic bronchitis and asthma.

Rachitic Deformities of the Chest.—As the result of softening of the bones due to rickets in early childhood, with or without the effects of respiratory diseases causing obstruction to inspiration, three varieties of rachitic thorax are met with, namely: (1) The *simple rachitic chest,* (2) the *"pigeon-breast,"* (3) the *transversely constricted chest.*

(1) Simple Rachitic Chest.—This type of chest is shorter and deeper than normal. It is characterized by a shallow depression or groove on either side, running downward and outward in about the anterior axillary line, causing the anterolateral portions to sink in and the anteromedian to project forward, the softened, rickety ribs yielding (principally at the costochondral junctions), owing to lessened intrathoracic pressure during inspiration. As shown in Fig. 13, the nearly circular outline of the healthy child's chest becomes almost quandrilateral in the rickety. Another characteristic is the "rachitic rosary," or beadlike enlargements of the cartilaginous (sternal) ends of the ribs, sometimes easier felt than seen.

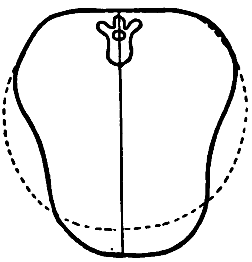


Fig. 13.—Simple Rachitic Chest. Dotted line indicates the shape of the chest in an infant about same age.

(2) Pigeon Chest.—The sides of the chest are flattened and the sternum pushed and arched forward so that the transverse outline is triangular, with rounded sides and angles (see Fig. 14). The protrusion of the sternum is most marked at its lower portion, the ribs sloping sharply backward as the bow ribs of a boat bend from its keel

(*pectus carinatum*). This exaggerated rachitic deformity occurs after diseases in which great strain is thrown upon the lung by obstruction to inspiration, as from whooping-cough, protracted bronchial catarrh, nasal, pharyngeal, or faucial enlargements (turbinates, adenoids, tonsils). The external atmospheric pressure thus overbalancing the internal, the plastic ribs yield inward at their least resistant portions. Indeed, the pigeon-breast may result from such affections in infants who may not be rickety, especially with the prolonged cases of bronchopneumonia following measles or pertussis.

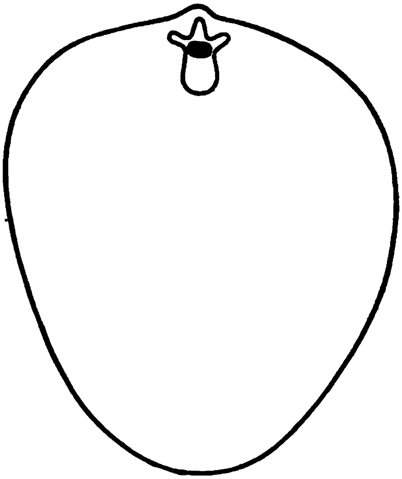


FIG. 14.—TRANSVERSE SECTION OF A RACHITIC CHEST AT LEVEL OF SIXTH THORACIC VERTEBRA. Circumference, 32½ inches; right half, 16½ inches; expansion, 2 inches. (After Musser.)

(3) TRANSVERSELY CONSTRICTED CHEST, OR HARRISON'S SULCUS.—In this form the lower ribs, corresponding to the diaphragmatic attachment, are drawn in below the sterno-xiphoid junction, downward and outward as far as the axilla. Harrison's groove is produced by the same causes as for pigeon-chest, and the two deformities are frequently associated. Long-continued and hard coughing and inspiratory difficulty, by increasing the pull of the diaphragm upon the flexible, softened ribs, cause the deepest depression just above the upper limits of the liver and spleen, the resistance of the latter often making the lower margin of the ribs flare out. This expansion of the lower opening of the thorax may be the result, however, of pressure from gaseous distention of the bowels (acute cases), or from abdominal dropsy and large tumors. Again, it is seen in some healthy, large-chested adults.

The significance of the detection of the rachitic types of chest lies in directing attention to the probable evidences of the causes mentioned, and of the respiratory incapacity preceding and accompanying pulmonary tuberculosis.

Funnel Chest (Trichterbrust).—This consists in a marked depression of the lower part of the sternum, the outer border of the "funnel" being as much as 3 or 4 in. in diameter, and the apex from 1½ to 3 in. deep. It is usually congenital, and may be associated with other physical and mental signs of hereditary or developmental faults and degeneration. It also may be of rachitic origin.

A sort of acquired funnel chest, with a sinking-in of the ensiform cartilage chiefly, is seen occasionally in cobblers and carpenters who in early adolescence began to work at their trades, and for many years used tools with considerable pressure over that region. The respiration and cardiac action are likely to be interfered with only in the congenital variety.

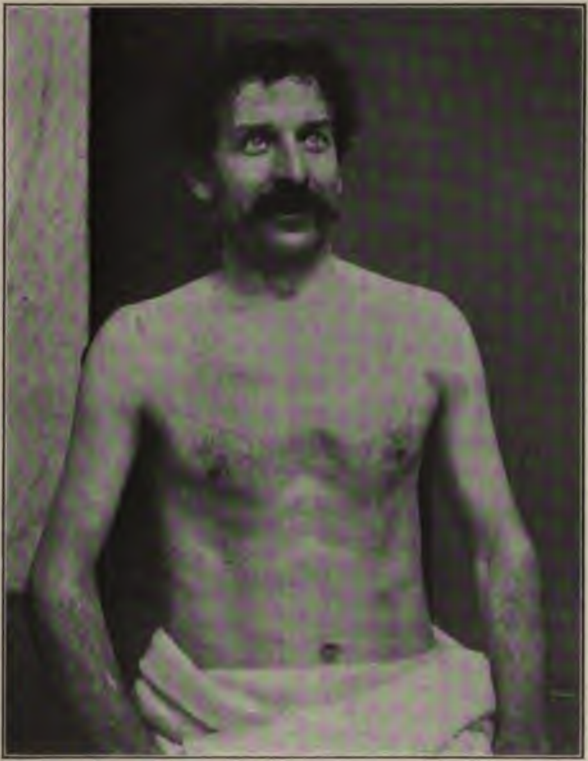


FIG. 15.—FUNNEL BREAST.

(3) SYMMETRY

Irregular Deformities of the Chest.—These are caused by the various *spinal curvatures*; inspection of the back may reveal a *kyphosis*, or bending backward of the spine; or a *scoliosis*, or bending sidewise;

or, more likely, a *kyphoscoliosis*, or combination of both—i. e., a simultaneous lateral and posterior curvature; more rarely, a bending forward, or *lordosis*, may be met with. The kyphoscoliotic chest witnesses the most distorting alterations of size, shape, and symmetry at once. The spine is twisted; the upper dorsal region is humped; one shoulder is lower and perhaps more posterior than the other; one side is irregularly reduced and retracted as compared with the other, and the ribs and other thoracic bones are distorted and out of relation, so that the landmarks and topographic relations of the likewise compressed and displaced thoracic organs are without value in diagnosis. Incidentally, Pott's disease of the spine may be discovered, as well as abnormal rigidity (*spondylitis deformans*) of the vertebræ.

Chest deformities from curvatures of the spine are indicative of susceptibility to, and often the presence of, tuberculosis, unilateral or local emphysema, pneumonitis, atelectatic lung, pleuritis, and degeneration and dilation of the myocardium, especially the right ventricular wall.

Unilateral Abnormalities of Size and Shape.—(1) UNILATERAL CONTRACTION of the chest is noted by observing (*a*) the general drawing-in and flattening on one side; (*b*) the intercostal spaces are both narrowed and depressed, and the ribs may be so close together, especially at the lower lateral regions, as to overlap as do shingles; (*c*) the shoulder on that side droops; (*d*) the semicircumference is visibly and measurably diminished, while the unaffected side is slightly increased owing to compensatory expansion; (*e*) the mamma and scapula are nearer the median line; (*f*) the spine is curved, with its convexity toward the sound side. This asymmetry is caused by a

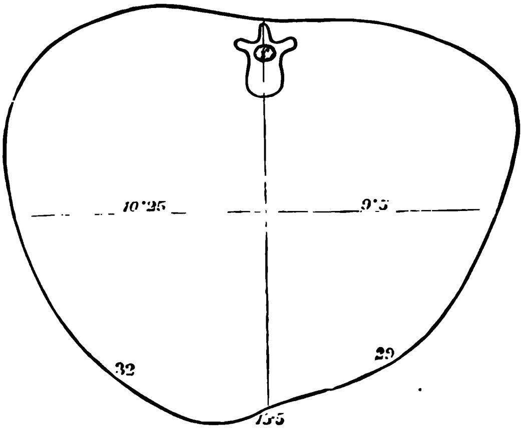


FIG. 16.—UNILATERAL RETRACTION OF CHEST, CONSEQUENT UPON CIRRHOSIS OF LEFT LUNG, IN A GIRL OF FOURTEEN YEARS. The figures indicate anteroposterior and transverse diameters and semicircumferences of right and left half of chest. (Gee.)

reduction in the size of the lung either from collapse or consolidation of the vesicles; thus it frequently follows long-continued pressure by pleural effusions, and even after the absorption or surgical aspiration of the liquid, compression of the lung is often kept up by chronic pleuritic adhesions; cirrhotic contraction of lung, or fibroid phthisis or interstitial pneumonia, as it is variously termed, is a cause second in frequency, the connective-tissue thickening not only preventing the lung from expanding with inspiration, but, when associated with pleuritic adhesions, actually drawing in the chest wall by the scarlike contraction. This inward traction pulls upon the heart and diaphragm also, with corresponding dislocation, and decreased freedom of function. Less commonly, unilateral shrinking may be the result of pulmonary collapse due to some such cause of a bronchial obstruction, as a foreign body within or a pressing mediastinal or pulmonary tumor from without, although a malignant growth may at the same time extend into and occlude the lumen of a bronchus.

(2) Unilateral enlargement of the chest has characteristics respectively the opposite of one-sided flattening—i. e., there is distinct general fulness or bulging on the affected side, seen particularly from the front; the intercostal spaces are less depressed, and, as in empyema, may be decidedly prominent, especially at the base, and widened also; the shoulder may be slightly elevated; the ribs may project outward a little with their inferior borders; the nipple and scapula are farther removed from the median line; the spinal bend, though slight, has its convexity directed toward the diseased side (the *larger*, as is the sound side with opposite retraction). While the semicircum-

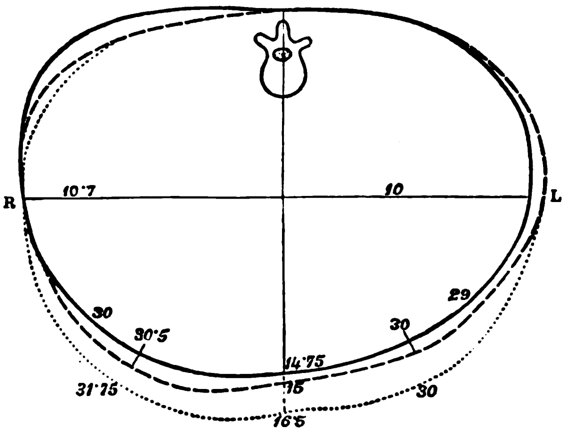


Fig. 17.—Unilateral Enlargement of Chest (Right Side), Artificially Produced by Injecting Air into the Right Pleural Cavity. Unbroken line: outline before injection. Broken line: outline after moderate distention. Dotted line: outline after extreme distention. Figures at bottom of vertical line indicate the anteroposterior diameter; along horizontal line, transverse semidiameter; remaining figures, right and left semicircumferences. (Gee.)

ference measures larger on the expanded side, there is simultaneousl some increase on the unaffected side, though relatively less, on account of the pathologic expanding force encroaching indirectly upo the healthy lung (displaced heart), and so causing it to exert greate compensatory effort.

One-sided prominence of the thorax is very seldom the effect o a " vicarious emphysema " or dilation of the vesicles, because of disease of the opposite lung. More frequently it is produced by distention with gas or liquid in the pleural sac; pneumothorax, variou pleural effusions. Slight unilateral fulness may be seen in cases o pneumonitis affecting the whole lung. Great disparity between th two sides is readily observed in certain conditions in which eithe side is enlarged and the other contracted.

An occasional *apparent* symmetry of both sides of the chest i found even in the presence of a large pleural effusion on one side due to the fact that the compensatory expansion on the unaffected sid balances the enlargement on the diseased side.

Local Irregularities of Form.—(1) LOCAL OR CIRCUMSCRIBED DEPRESSIONS of the thorax are most frequently noticed in the supra and infraclavicular regions, caused by apical consolidations or cavities (tubercular), or localized pleuritis. Extreme care must be exercised that a slight flattening near the clavicle is not adjudged to b from tuberculosis when an unsymmetrical position of one arm o shoulder may be the cause. A deepening on one side of Morenheim' depression, at the outer part of the infraclavicular region, is a sig of phthisis. Flattenings or depressions may be seen over any part o the lungs—anteriorly, laterally, or posteriorly—that may be the sea of phthisical or bronchiectatic cavities, circumscribed pleuritic adh sions, abscesses and gangrene of the lung. Two sources of erro should be guarded against: apparent depressions of the chest due t muscular wasting; relative deepening of the supra- and infraclavicula spaces by the projection of deformed clavicles, the result of heale fractures. Finally, a partial shrinking may be seen, often posteriorl at the lower ribs after the absorption of a small pleuritic exudatio

(2) LOCAL OR CIRCUMSCRIBED BULGINGS of the thorax are of comparatively frequent occurrence, particularly in the cardiac and aorti regions; in regard to the latter, consideration will be given und the section dealing with heart affections—with aneurism and cardia enlargements, for example.

In the *first* place, local bulgings may be due to pathologic cond tions in the *chest wall*—congenital or developmental irregularities i the formation of the ribs, costal cartilages, and their junctions, as from spinal and rachitic deformities; periostitis and abscesses of the subcutaneous cellular tissue; enchondroses and actinomycosis of the ribs; lipomatous, sarcomatous, carcinomatous, and gummatous *tumors* of the chest wall or of the lungs, pleuræ, mediastinum, or bronchial glands eroding and pushing their way through the wall. *Secondly,* from within, localized emphysema, cutaneous or pulmonary or local protrusions of a general emphysema may be seen in the supraclavicular regions or along the upper anterior borders of the lungs; at the base of the chest a localized prominence, with several smoothed-out interspaces and edematous skin, the pointing of a pleural abscess (empyema), may be met with; in or near the same region, encapsulated pleuritic exudations or a circumscribed pneumothorax may exist; a little lower the bulging of a subphrenic abscess may appear; finally, in rare cases, hydatids and hernia of the lung may be thought of.

It should be noted that prominences due to pleural and pericardial effusions are much more apt to show distinctly in children than in adults, on account of the greater flexibility of the ribs in the former, so that the absence of marked bulging in the latter does not preclude the possibility of a large exudation where the walls are more rigid.

Care must be exercised also not to give pathologic significance to the perfectly natural slight prominences at the right back inferiorly, and the precordial region in many children and some young adults, quite apart from the asymmetries of muscular development, especially anteriorly.

(4) RESPIRATORY MOVEMENTS

(1) **Pathologic Alterations (Bilateral or Symmetrical) of the Type of Respiration.**—(*a*) INCREASED EXPANSION (predominance of the *thoracic* or *upper costal* type of women).—Whenever exaggerated thoracic movement is observed (the costal type in men, its excess in women), the first point in the analysis is the proving of its physical genuineness—i. e., the exclusion of voluntary forced breathing and of hysteria. Briefly, all conditions which impede the action of the diaphragm cause a relative increase in the thoracic type of movement, and a corresponding decrease in the inspiratory protrusion of the upper abdomen.

Such diaphragmatic restriction is characteristically seen in cases of inflammation of the pleural and peritoneal serous membranes coating the diaphragm (diaphragmatic pleurisy; general and local peri-

tonitis), and of mechanical restraint due to increased upward pres
within the abdomen. Among the principal causes of the latter
gaseous distention of the intestines, ascites, and great enlargem
of the liver or spleen, or other abdominal tumors; advanced p
nancy is a frequent cause. Besides these, the weight of a large p
cardial effusion may prevent free action of the diaphragm, and t
cause exaggerated thoracic movement; sometimes the failure of
diaphragm to act on the left side in such cases can be detected
noticing the sucking-in of the lower interspaces with inspiration
that side. Again, complete absence of costo-abdominal moven
from paralysis of the diaphragm, the result of a severe diffuse p
tonitis or of a bulbar palsy or of paralysis of the phrenic nerves oc
ring with multiple neuritis or spinal pachymeningitis; here the up
costal movement is excessive, and instead of epigastric protru
during inspiration there is general retraction, followed by appa
protrusion with expiration as the chest wall recoils.

(*b*) DIMINISHED EXPANSION, on the other hand, with exagger
abdominal respiratory movement, may be a simple or direct diminu
of simultaneous expansion and elevation, or the diminution may be
to a modified relation of the movement of expansion to that of el
tion, causing a changed character of thoracic breathing. Insta
of the former are observed in lack of development, ordinary debil
paralysis of the chest muscles, tuberculosis of the lungs, tetanic sp
of the chest muscles, whether from disease (tetanus) or strych
poisoning; rarely, in association with the conditions of rigidity
thickening of the thorax due to myositis ossificans and sclerodern

General diminished expansion may also be an indication of par
or complete obstruction to the entrance of air into the lungs. '
causes of this condition may reside in (1) the larynx, trachea,
larger bronchi; (2) the smaller bronchi; or (3) the pulmonary v
cles. Severe spasmodic or membranous laryngitis, paralysis of
vocal cords, laryngeal tumors, acute laryngotracheitis and bronch
and the pressure of tumors from without these upper respiratory
sages, are examples of the first. Under the second, spasmodic
bronchial asthma, and the deep-seated bronchitis of influenza, o
frequently seen in the two extremes of life, are common examp
Obstructions occurring under the third head may be either withi
without the lung vesicles; of the former, we have double or bilat
pneumonic consolidation, tuberculosis, and pulmonary collapse
causes; of the latter, double hydrothorax. Besides these, it sh
be noted that the general thoracic movement may be diminishe

the result of pain during breathing in cases of acute pleuritis, pleurodynia, or intercostal neuralgia.

Again, in the *emphysematous chest* the normal respiratory movement is diminished because characteristically modified or replaced by an up-and-down heaving of the thorax as a whole. It is here that the relation of expansion to that of elevation is so altered that the former is practically in abeyance, and the rigid chest moves vertically only. This abnormity of movement depends upon the fact that the lungs are constantly overdistended with air because the vesicles have lost their elasticity, and hence their expulsive force. The air-filled vesicles permit of no further expansion, therefore the forced effort to do so produces simply an exaggeration of the mere dragging upward of the thorax seen in normal forced inspiration. "The inspiratory movement must be made 'on the top of inspiration'; that is, *plus* a degree of inflation of the lungs that in health should be approached only toward the *end* of inspiration" (Steell).

We observe simultaneously with the upward movement of the upper part of the chest that there is anterolateral retraction of the epigastric and lower costal regions during the inspiratory effort, owing to the forcible descent of the diaphragm not being followed by the expansion of the lower lobes of the lungs, and the external atmospheric pressure thus overcoming the weaker internal.

Expiratory chest movement is attended with even greater difficulty (dyspnea) than the inspiratory; it is imperfect, slow, and extremely prolonged. Obviously this is because of the diminished elasticity of the lungs, aided in many cases by obstruction to the egress of air from bronchitic exudation in the tubes, and sometimes by downward displacement of the diaphragm.

(*c*) RECESSION OF THE LOWER, ANTEROLATERAL INTERCOSTAL SPACES during inspiration, instead of expansion, is characteristic of obstruction of the upper respiratory passages, especially of the larynx, as from spasm of the glottis, edema of the larynx, diphtheritic laryngitis, and the like. This inspiratory retraction of the lower zone of the chest is most manifest in children, where the upper epigastric region, as well as the interspaces, may be drawn in considerably from obstruction above the bifurcation of the trachea. The inspiratory expansion of the upper thorax is forcibly made for even the little air that finds its way into the upper lobes of the lungs; diaphragmatic contraction is also greatly intensified, with marked abdominal protrusion, but as there is not sufficient air to inflate the lower lobes, their collapse allows the atmospheric pressure to drive in the inferior parts of the chest. This may be witnessed in extensive bronchopneumonitis of both lungs, as well as in emphysema, mentioned under the previous heading; and it is the prolonged strain of the persistence of the former of these two in children with rachitic, rib-yielding chests that leads to the permanent transversely constricted and pigeon chest.

(2) **Unilateral Abnormities of Movement.**—UNILATERAL DIMINUTION OF MOVEMENT is best exemplified in large pleuritic effusions on one side. The abeyance of movement may be associated with simultaneous unilateral enlargement or retraction of the chest, or without any visible change to either abnormity. Diminished expansion, or even virtual absence of one-sided movement, with abnormal fulness on that side, is seen in extensive pleural exudations and in pneumothorax, and slightly in massive pneumonic consolidation. The principal causes of unilateral decreased movement with retraction of the affected side are fibroid phthisis, partial or complete collapse of lung from old pleuritic adhesions or from pressure on a main bronchus by a tumor or aneurism. In cases of acute pleuritis in the early stage (before liquid effusion) restriction of movement on one side without any alteration of its size or shape is usually met with on account of the nervous reflex influence of the pain of breathing; also in cancerous infiltration of the lung.

What slight movement may exist with the unilateral diminution is that of elevation rather than of expansion, such as difficult respiration generally produces. The opposite or unaffected side usually shows compensatory increase of action.

UNILATERAL INCREASED EXPANSION, as just stated, is a signal indication of incapacitated or diseased lung on the other side, and consequently represents vicarious action, or "compensatory emphysema," from the temporarily exaggerated distention of the pulmonary vesicles. The contrast between the overacting healthy and under- or nonacting diseased side is the more readily detected by noting the sharp inspiratory onset, and more rapid and vigorous movement of the former.

(3) **Local Abnormities of Movement.**—LOCAL DIMINUTION OF MOVEMENT is most characteristically observed in the lagging breathing of tubercular consolidation of the apices of the lungs, especially in the infraclavicular depressions. The indication of this condition on either side is a delayed as well as diminished inspiratory movement. Local flattening or sinking-in accompanies the lagging deficiency of movement; this is true of localized pleurisies (adhesive)

as well as phthisical consolidations of the upper lobes. Sometimes a tuberculous cavity may be diagnosed by noting a slight trembling or flapping of the thin overlying intercostal tissues in emaciated subjects. Circumscribed diminution of expansion of the upper thorax may also be due to apical pneumonitis. At the base, with some fulness of the interspaces the deficient movement may be caused by lobar pneumonitis or a small quantity of pleural effusion. In children suffering from bronchopneumonitis, small portions of consolidated and collapsed (atelectatic) lung may be localized by the discovery of such little areas of defective movement.

LOCAL INCREASED MOVEMENT is compensatory to circumscribed impaired action on the same side; thus, an increased expansion of the upper part of the chest on the right side, with restriction at the base, is an indication of pneumonic consolidation of the right lower lobe, or possibly of a pleural effusion. Again, when the apex of a lung is the seat of a tuberculous infiltration, the respiratory movement of the lower part of the affected side is visibly increased.

ABNORMAL PULSATIONS AND RESPIRATORY BULGINGS.—These are likewise local physical signs. Rarely, *pulsations of the heart* may be seen in the left mammary or axillary regions due to overlying infiltrated lung or an accumulation of pleuritic pus (*pulsating empyema*), to and through which the cardiac impulse is transmitted. Such communicated pulsations are to be differentiated from hypertrophy, dislocation, and aneurism of the heart and aneurism of the descending aorta.

Inspiratory bulging may be seen above the clavicles and near the upper edges of the sternum in considerable vesicular enlargement of emphysema.

Expiratory bulging of the supraclavicular and intercostal spaces is more common in emphysema and asthma on account of the difficulty in emptying the lungs during the costal depression; in cases of large lung cavities, the walls of which are adherent to the chest wall, bulging of the upper interspaces during expiration, especially when forced, may also be observed in advanced tuberculosis.

Finally, an *inspiratory fall* and an *expiratory rise* in the bulging of the lower interspaces may sometimes be seen as an index of the weakening of the intercostal muscles by the accumulation of a sufficiently great quantity of liquid in the pleural sac, or the pleura and muscles may even be ruptured so that the swelling is quite subcutaneous.

Circumscribed thoracic pulsation may be due to a tumor in proximity to the heart. Sailer describes a case of distinct expansile pulsation over the whole left thorax which autopsy showed to be caused by a ruptured aneurism of the ascending and transverse portions of the arch of the aorta, with left hemothorax.

Stiller's Intercostal Phonation Phenomenon.—The simultaneous bulging of the intercostal spaces during phonation is held by Stiller to be a useful adjunct in the physical diagnosis of a pleural effusion and its differentiation from consolidation of the lung. The phenomenon also enables one to trace the line of demarcation of liver and spleen. It is best elicited if the patient closes his nostrils and places his hand over the mouth to partially obstruct the expired air. Forced expiration should also be practised before phonation is attempted (Abrams).

(4) **Degree of Respiratory Expansion.**—In doubtful instances of bilateral, unilateral, or local alterations of thoracic movement, or whenever inspection requires the aid of additional accuracy, mensuration is resorted to, and the precise *degree* of expansion compared with the normal. If the amount of expansion is less than 2 in. in the male and 2½ in. in the female, it is below the normal average respectively.

An expansion of but 1½ in. in a large chest in one who has or has had frequent attacks of asthma or bronchitis, points to the development of emphysema; in marked cases the diminution may be to even ½ in. In small chests, as the phthisical, the degree of expansion varies from 1 to 2 in. in most of the cases.

Semicircumferential diminution of expansion is noted over pleural effusions, pneumothorax, chronic pleuritic adhesions, and fibroid shrinking of the lung. In acute pleuritis and lobar pneumonitis there is reduced measurement, almost to a temporary abolition of motion, on account of intense pain and mechanical interference causing partly reflex and partly voluntary restriction.

Local diminution of expansion is seldom a mensurable factor of importance except in occasional cases of early pulmonary tuberculosis. Here observation may be aided by measuring the anteroposterior apical expansion on both sides with the thoracometer, one arm of which is applied below the middle of the clavicle and the other upon the spine of the scapula, care being taken that both extremities of the instrument are equidistant from the median line of the chest. In a case of apical consolidation or excavation on one side, for example, the diminished respiratory excursion may readily be measured, so that the forward movement on the affected side during quiet breathing may be abolished or scarcely perceptible on the scale, instead of indicating from $\frac{1}{60}$ to $\frac{1}{14}$ of an inch (Sibson), or, during forced inspiration, may register decidedly less than ½ to 2 in.

(5) **Abnormal Vital Capacity.**—Mensuration of the capacity for air by means of the spirometer, although not of absolute value in diagnosis, may be of considerable service in watching the course of a given case of pulmonary affection by taking frequent measurements, and so better estimating the probable outcome; it supplements the other methods of physical examination in comparing present with past data; also affords indications of the general soundness and enduring qualities of groups of men, such as those about to enlist in the army and navy, or candidates for gymnastic contests or field and aquatic sports. In view of the many conditions, including anatomical and physiological peculiarities, habits, occupation, the state of the abdominal organs, etc., that may affect the vital capacity aside from thoracic disease, the best available criterion is its relation to body height. Otis and Hitchcock found in 24,000 measurements an average lung capacity in general of about 236 cu. in. The average lung capacity of a young male adult 5 ft. 7 or 8 in. in height is 237.10 cu. in.; that of one 5 ft. 8 or 9 in., 244.44 cu. in.; one 5 ft. 10 or 11 in., 261.38 cu. in. The general average is 3½ cu. in. for each inch of height, or 23.19 c.c. for each centimeter of height. In women, the average capacity for average height is from 145 to 150 cu. in., or 2.3 cu. in. for each inch of stature.

Any decided fall below the general average of lung capacity justifies a suspicion of tuberculosis, though it does not warrant a positive diagnosis as an independent observation. In fact, the spirometer indicates only the deficient supply of air without really pointing out which of many causes produces the deficiency, or in what portion of the lungs the deficiency is located. Whether the diminution of vital capacity is due to weak or diseased lungs or to the fallacious results of ignorant or intentional incoordination of breathing in making the test in certain individuals, the need for instructions in respiratory gymnastics in either case is clearly indicated. It should be borne in mind that a convalescing patient increases in vital capacity with practise as well as with the general course of improvement. The degree of diminution of spirometric measurement varies, of course, with the extent and intensity of the pathologic interference with the lung action; that is, whether the disease is narrowly circumscribed, one-sided, or bilateral, and whether it is severely acute and rapid in course, like lobar pneumonitis, or the reverse, like emphysema.

(6) The degree of respiratory pressure, as obtained by the pneumatometer, is so closely related to that of expansion that brief reference to it may be made here. Its results are more variable than those obtained by spirometry; but they have auxiliary value, nevertheless, in estimating either the severity of disease or its tendency to improvement or aggravation. In health, the expiratory pressure, owing to the muscular force that may be exerted, is greater than the inspiratory in about the ratio of 7½ to 5.

Diminution of inspiratory pressure in stenosis of the respiratory passages and in pleuritis with fibrinous or liquid exudation (from pain or mechanical interference) are naturally concomitant. In pulmonary tuberculosis, especially in the earlier stages, the progress of the disease may be watched to advantage by noting the changes in the diminished pressure of inspiration.

Of more diagnostic significance, however, is diminished expiratory pressure, observed in emphysema, where the degree of diminution measures the degree of loss of elasticity and tension of the vesicles of the lung.

(5) LITTEN'S SIGN

As intimated before, we have in the observation of this sign of the moving phrenic shadow a ready and fairly reliable means of judging of pathologic diminutions of respiratory capacity without apparatus.

Bilateral decrease of the moving phenomenon, and consequently of the downward propulsion of the diaphragm may mean either a tubercular predisposition, or actual disease, or moderate emphysema; if the former, the characteristic small or phthisical chest is associated, and if the latter, the enlarged, barrel-shaped chest. Great general debility and muscular weakness from prolonged illness of various kinds may also manifest diminished but not absent excursion of the shadow.

One-sided diminution of the diaphragm shadow may mean small pleuritic effusion, pneumonia of the lower lobe, or pleuritic adhesions, the effect being to lower the point at which the shadow begins to move by an interspace or two. The special value of Litten's sign is in the differential diagnosis between a liquid pleural effusion on the right side and a subdiaphragmatic abscess or upward enlargement of the liver; the absence of the phenomenon in the first-named condition is thus distinctly helpful, being especially indicative of large fluid accumulations.

Only very large tumors below the diaphragm or very large quantities of ascitic fluid interfere sufficiently with the descent of the diaphragm as to abolish the moving shadow.

(6) ABNORMAL RATE AND RHYTHM OF RESPIRATION—DYSPNEA

Alterations of the frequency and regularity of the breathing movements give rise to more or less *dyspnea*, or "shortness of breath," at once an objective as well as subjective symptom, and here considered because closely associated with the physical signs proper of thoracic diseases, although it may be caused by many conditions outside of the chest. Pathologic dyspnea is the counterpart of the physiologic shortness of breath produced by intense exertion, as a sharp run up-stairs, or by fulness of the stomach after a too convivial meal; it is an abnormal increase of respiratory activity.

Anomalies of Frequency.—SIMPLE ACCELERATION of the respiratory rate is a feature observed in almost every variety of disease of the respiratory organs; at the same time, it should be noted that laborious breathing, or true dyspnea, is also nearly always increased in frequency. Although abnormally rapid breathing is secondarily suggestive of other than pulmonary conditions, such as various febrile states—especially in children—hysteria, toxemic conditions, anemia, certain cardiac diseases, etc., these are more appropriately dealt with in works on general medical diagnosis.

Anything which completely or partially obstructs the flow of respiratory air may accelerate the breathing. Thoracic conditions causing painful breathing, of course, may cause abnormally rapid respiration independent of much mechanical interference with the flow of tidal air, or may aggravate it. Thus, inflammation of the pulmonary, costal, or diaphragmatic pleura, with or without pneumonitis, is a common example of *hyperpnea*, as abnormally frequent respiration is sometimes termed. The respiratory affections that give rise to acceleration of breathing movements likewise produce dyspnea, and will be enumerated later.

ABNORMALLY SLOW RESPIRATION (*hypopnea*) is usually associated with stenosis of the larynx, as from tumors within or pressing from without, foreign bodies in the trachea, and so on. Again, even in cases where the air-space in the pulmonary vesicles is diminished in some portions, provided the interference with respiration is gradually produced by a chronic process, slowing of the breathing is more characteristic than rapidity, the patient being at rest. This is seen in chronic fibroid phthisis.

Anomalies of Rhythm.—(1) SIGHING, or the occasional deep inspiration followed by a rapid and broken expiration, is a temporary irregularity of breathing often met with in healthy persons, sitting or sleeping, but who are not inhaling sufficient oxygen constantly. It indicates at times deficient respiratory capacity from habits of poor expansion due to sedentary, stooping occupations and neglect of corrective exercise.

(2) SIMPLE IRREGULARITY.—There are several varieties of irregularity of rhythm met with. Thus, the respiratory movements may be (*a*) abnormally shallow and irregular in the time intervals; (*b*) abnormally deep and irregular; (*c*) spasmodic, jerky, and restrained The first is seen in conditions of collapse, as in the terminal stages of pulmonary tuberculosis. The second, besides being sometimes associated with the abnormally full breathing in certain diabetics—the so-called *diabetic dyspnea*—is not infrequently observed in patients suffering from acute miliary tuberculosis of the lungs. The third is most commonly *inspiratory*. It is especially characteristic in the "catch" or "stitch" or "hot needle thrust" in the side of a beginning or dry pleuritis, acute pneumonitis, pleurodynia, or intercostal neuralgia. Instead of a continuous movement, the pain causes a sudden interruption or several interruptions of the inspiration which are easily noticeable; they may also be evident in asthma and in hysteria. Jerky *expiration* is seen in some cases of pleurodynia and pleuritis, also, as well as from fractured ribs.

(3) CHANGED RELATION OF INSPIRATION-EXPIRATION RATIO ASTHMATIC BREATHING.—A *shortened inspiration* is often characteristic of acute pneumonitis, and thus causes a *lengthening* of the *pause*, just before the expiration, that may be quite decided.

The difficult breathing of an asthmatic attack is marked by a reversal of the normal rhythm, so that the *expiration* is *longer* instead of shorter than the inspiration. The expiratory movement of the emphysematous chest is also relatively prolonged, but there is no the exaggerated use of the accessory muscles of respiration seen during an asthmatic paroxysm.

Increased length of inspiration is manifest in obstruction of th larynx or trachea; recession of the lower intercostal spaces and increased expansion of the upper thorax accompany this sign.

(4) CHEYNE-STOKES BREATHING, although associated more frequently with cerebral and renal than cardiac and pulmonary affec

tions, is always a unique and seriously significant alteration of the respiratory rhythm. The respiratory movements become gradually slower and shallower until a period of cessation or apnea ensues. This is followed by a very feeble respiration, then more quickly a stronger one, the succeeding movements becoming gradually deeper and quicker and noisier until pronouncedly dyspneic in character. These exaggerated respirations then decline in amplitude and rapidity again as before. The whole cycle of ascending and descending phases and pause lasts from a half to two minutes, the pause occupying about half the period. During this apneic pause consciousness is nearly or quite abolished, and the pupils are contracted and immobile to light; with the return of deeper breathing consciousness returns and the pupils begin to dilate and react to light. At the height of the regular dyspneic, deeply sighing, or snoring breathing the pupils may be wildly dilated.

Cheyne-Stokes respiration is usually more noticeable when the patient is quietly sleeping or comatose. There are several minor modifications of the type; thus, ordinary deep respirations may simply alternate with a regularly recurring apnœa, or the recurrences of superficial and exaggerated breathing may be minus the periods of pause.

Dyspnea.—Here belongs a further and more definite reference to difficult or laborious breathing, to its physical varieties and their causation; for, as just indicated, the chief characteristic of dyspnea is an increase in the frequency and depth of the respirations, or both, anomalies of rate and of rhythm.

INSPIRATORY dyspnea (meaning purely or dominantly a difficult entrance of air) is recognized by the increased energy and rapidity of the inspirations. They may be aggravated and interrupted by pauses to recover the breath on account of the least exertion, such as speaking, sitting up, etc. In severe cases the accessory inspiratory muscles are brought into active, almost tetanic contraction (the sternocleidomastoid, scaleni, pectoralis major and minor, the levatores costarum, serratus posticus superior, rhomboideus, trapezius, and levatores scapulæ). In various degrees it is the result of some obstruction in or about the upper respiratory passages. It is present in paralysis of the posterior crico-arytenoid muscles (dilators of the glottis), in spasmodic and membranous croup and edema of the larynx, in which the characteristic harsh, shrill, whistling, screeching, crowing, or barking inspiratory sound is heard; it is known also as *stridulous breathing*, or *stridor*. Similarly, laryngeal and tracheal obstruction from foreign bodies (dyspnea sudden) and tumors within, and pressure from the latter without, produce inspiratory dyspnea. In extreme cases there is usually at the same time inspiratory retraction of the lower interspaces and epigastrium, and even the upper and supraclavicular spaces.

The croupous bronchitis and bronchopneumonitis of children give a typical picture of inspiratory dyspnea.

Similar results happen when conditions arise suddenly or gradually that diminish the air-space or disturb the "volumetric variation" of the lungs. Thus, perforation of the pleura over a tuberculous cavity, and consequent collapse of the lung from pneumothorax; sudden or rapid congestion or edema of the lungs, or effusion into the pleural sac; the occluding pressure of aneurismal and mediastinal tumors; thrombosis and embolism of the pulmonary artery; kyphoscoliotic deformity of the chest, interfering with the extent of breathing surface, and abdominal enlargements which push up the diaphragm considerably.

Expiratory dyspnea is characterized by a prolonged, labored, often wheezy expiration. The abdominal muscles, the quadratus lumborum, and the serratus posticus inferior are accessory muscles. This variety of dyspnea may be caused by movable tumors obstructing the trachea and bronchi, but is due principally to asthma and emphysema.

Mixed inspiratory and expiratory dyspnea is very frequent, and occurs in most of the respiratory and cardiac affections at some time or other.

Orthopnea (Gr., *orthos*, straight, upright) is an exaggerated form of dyspnea requiring an upright posture for the purpose of getting the breath. The accessory muscles of respiration are strongly active, and the short, quick, heaving efforts witness to the gravity and distress of the condition. The sitting or standing position enables the patient's thorax to move with the greatest freedom, and the auxiliary muscles to act better. Orthopnea is a usual sign of cardiac disease in the advanced stages, when the "broken compensation" on the part of the muscular walls of the heart results in pulmonary congestion, and later in dropsy of the lower extremities and congestion of the abdominal viscera. In bad cases of asthma and emphysema, also, and in severe fibrinous bronchitis, bilateral pleural effusions, and double pneumonites, the up-and-down instead of the expansile movement of the chest in the sitting posture is a common observation, especially in the medical wards of a large hospital. In cardiac orthopnea, the in-and-out movements of the ribs, though exaggerated, are much better maintained than in the respiratory form

CHAPTER III

PALPATION

To learn more about the physical characteristics of what one sees, one instinctively and almost simultaneously puts forth the hands to feel whether vision has been true and sufficient or incomplete and inexact. Naturally, then, the method of trained touch, or *palpation*, comes close upon the heels of inspection to corroborate, modify, and enlarge the knowledge gained by the latter. However, a sensitive, educated touch is not only useful in aiding the sense of sight, by confirming and amplifying the results of inspection; it has independent value, also, in furnishing decisive evidence that even the more exact methods of percussion and auscultation must sometimes wait upon, as in the relation of the vocal fremitus to differential diagnosis.

On account of the ease and simplicity with which palpation may be performed, beginners are prone to neglect its careful cultivation. This is a mistake. The tactile sense cannot be practised too much or too precisely. It often happens that, for various reasons, as of prostration and exhaustion, or of delicacy of feeling on the part of the patient, inspection cannot be satisfactorily done, whereupon larger dependence must be placed on palpation for ascertaining the general results of the former method.

GENERAL METHOD OF PALPATION

As in inspection, the chest should be entirely or sufficiently bared so as to make the direct and comparative investigation of every region as definite as possible. Occasionally the hand may be slipped beneath the gauze underwear. The posture of the patient should be in a manner both even and easy, whether standing, sitting, or lying, avoiding especially asymmetries of contour due to a shrugged shoulder, turned neck, or improperly placed hands and arms.

Before applying the hands, the examiner should see that their palmar surfaces are warm and dry, and the nails as short as the fleshy finger tips; and the hands first, then the finger tips, if need for more accurate localization, may be employed by applying the gently and flatly, but firmly, upon the parts to be palpated; col clammy hands, sharp, scratchy finger nails, and an abrupt, rough, poking pressure are not only objectionable to the patient, but by t more or less nervous apprehensiveness or resentful resistance of t patient cause incoordination of movements and reflex or volunta rigidities that seriously interfere with the reliability of the physic signs. Always, corresponding regions on the two sides are to palpated by a simultaneous application of both hands, or by applyi one hand similarly on both sides, or by alternately pressing with ea hand on its respective side. Thus, the infraclavicular, axillary, a infrascapular regions, for example, are studied comparatively each case.

PALPATION OF CERTAIN INSPECTED SIGNS

As regards the general and unilateral abnormalities of size, shap and symmetry of the chest, palpation is hardly useful as an adjun to inspection; but in ascertaining the presence and characteristics local deformities, and also of local alterations of movement, th method is of considerable practical value.

Thus, palpation gives exactitude to the observations of bony prom nences, supraclavicular depressions, intercostal widenings, etc., f example.

Again, important aid is derived from palpation in the percepti of local *asymmetries of movement.* The infraclavicular regions a usually first examined by placing both hands there, the finger ti resting in the first intercostal spaces and pointing a little outward, that the hands form a V-shape. The axillary regions may be p pated standing either in front of or behind the patient; likewise wi the infrascapular regions, although, when standing behind, the patie should bend forward so as to permit the examiner to apply the han vertically and avoid the dubious results of an otherwise constrain position of the wrists.

Owing to the frequency with which that most prevalent and gra pulmonary disease—tuberculosis—attacks the apices of the lun especial care is needed in acquiring the best procedure and m delicate skill in the palpation of the supraclavicular and supraspino regions of the thorax, in order that the early evidences of the dise may be detected, since upon these do the prognosis and prompt a rational treatment depend. Standing in front of the patient, who may be seated, two or three finger tips are allowed to rest in the supraclavicular spaces, with the fingers gently curved very much after the manner of a pianist or typewriter. A little more flatly applied behind the anterior borders of the trapezius muscles, the fingers may here also discover one-sided abnormal movement. The detection here of delayed and diminished movement, "expiratory drag," or "lagging" breathing, is strong evidence presumptive and particular of the deficient expansion of pulmonary vesicles associated with incipient tuberculosis. Lagging, or less and later movement over the lower part of one side is usually indicative of pleuritis or pneumonitis, possibly of infarction or of pleurodynia on that side.

The freedom, extent, and symmetry of diaphragmatic movements may also be palpated by applying the hands (finger tips) over the epigastrium; the irregular, restricted, and one-sided movements of diaphragmatic pleuritis, phrenic nerve palsies, and local peritonitis, may thus be felt. Finally, palpation may be employed to aid inspection in noting the frequency and rhythm of the respiratory excursions.

THORACIC VIBRATIONS: VOCAL FREMITUS

NORMAL

When the hand is laid upon the chest of a healthy person while speaking, a fine, vibratory, tremor-like sensation, or whizzing, is felt. This voice vibration or resonant thrill is called the *vocal fremitus* or *tactile fremitus*, less commonly the pectoral fremitus. The sound vibrations of normal breathing, or the vesicular respiratory murmur as it is called, are appreciable only by the ear and not by the hands.

The *method of examining* the vocal fremitus is to have the patient utter certain words in distinct, even, measured monotones while the examiner's hands are applied upon different regions of the chest in a comparative manner. The old way is to ask the patient to repeat the count, "one, two, three." As the normal vocal vibration is delicate under all circumstances, and for this reason is easily deadened by too forcible pressure of the hand, it is more effective to have the patient use the words "ninety-nine," or such artificially phrased terms as "nom-nom-nom," by which the predominance of the nasal consonants adds resonance to the tones. To determine the finer vibrations, it is advisable to employ the tips of the first, second, and third fingers instead of the whole hand.

Besides the simple factors of the strength of the voice—i. e., its loudness, coarseness, and graveness—the intensity of the vocal fremitus depends upon several other physical conditions, as follows: (*a*) *On the pitch of the voice:* the lower the pitch the more marked the fremitus, and *vice versa.* This explains why the vibrations are usually more distinctly felt in adults than in children, and in males than in females. Furthermore, just as in musical (stringed) instruments the vibrations of the lower strings are much more perceptible than those of the upper strings, being of greater amplitude and fewer in number in a given time, so the fremitus of the singing or speaking voice in the bass, baritone, or contralto register is more marked than in the tenor or soprano, and accompanies the lower notes of any given register more than the upper; the thin, high-pitched, "throaty" notes are without fremitus. (*b*) *On the size or diameter of the bronchus* conducting the vocal vibrations, and its *nearness to the chest wall.* Hence the fremitus is more marked on the right side because of the larger, more superficial right bronchus. (*c*) *The thickness of the chest wall:* the thicker the chest the feebler the fremitus. The voice vibrations are much diminished or lost in passing from the elastic, air-containing lung tissue to the denser, more resistant muscle and fat. (*d*) *The distance of the part under examination from the larynx:* the greater the distance the weaker the fremitus. Therefore, the fremitus is more marked over the upper anterior aspect of the chest than over the axillary and infrascapular regions.

Normal Variations.—Because of the preceding physical and anatomical conditions, each individual will have his own degree of vocal fremitus or personal standard of normal fremitus, with regional variations dependent upon similar conditions. The relative intensities of fremitus due to these normal variations should be carefully noted, so that apparent exaggerations and diminutions of the fremitus are not erroneously considered pathological.

Normal right-sided increase of the vocal fremitus is the most important practical point to be borne in mind in this respect. This difference over the apices of the two lungs requires careful consideration in the diagnosis of beginning tuberculosis, because of the frequency of its occurrence here. This relative increase of the fremitus on the right side is explained by the slightly more massive and denser right lung, perhaps because of the firm, resisting liver beneath it, and by the additional fact of the anatomic relations of the right bronchus. Its largeness of lumen, shortness, and its leaving the trachea with such directness as the slightly acute angle gives it, permit greater force of vibrations to enter the right lung, and so transmit more sensation through the chest wall.

This slight physiologic increase of the vocal fremitus on the right side, as compared with the left, region for region, must constantly be borne in mind to avoid inferring a pathologic thickening of the lung on that side, or, on the other hand, some pleuritic encroachment upon the lung on the left side, because of the relatively diminished fremitus there.

Vice versa, the discovery of areas of diminished vocal fremitus on the right side, or of increased fremitus on the left, is naturally easier because of this normal difference noted just before, and all the more significant of morbid physical changes.

The borders of the lungs may be mapped out by the tactile fremitus in adults with strong, low-pitched monotones of the voice, especially the lower borders; the cessation of the fremitus as one reaches the liver on the right side is promptly ascertained.

Normal regional differences in the intensity of the fremitus, because of distances from the larynx, and variations in the thickness of the chest wall must also be allowed for. Therefore, the fremitus is more marked over the upper and anterior than the lower and posterior portions of the chest, being especially noticeable at the sternal halves of the infraclavicular regions and the interscapular regions. It is moderately felt at the axillary and infrascapular regions; but, owing to the thickness of the chest covering being greater on account of muscle, mammary gland, fat, and bone, the fremitus is considerably weakened at the mammary and scapular regions.

Moreover, in persons with thin-walled chests the fremitus is relatively much more perceptible in general, region for region, than where a large amount of dense tissue intervenes between the lungs and the palpating hands.

PATHOLOGIC

Pathologically, the vocal fremitus may be increased, diminished, or absent.

Increased Vocal Fremitus.—Generally speaking, all firm infiltrations of lung tissue cause increased fremitus, obeying the physical law that sound vibrations are conducted with greater intensity through solids than through air. Hence, bearing in mind the normal variations already noted, and estimating their relative prominence by a general survey in each individual examined, we proceed to localize any area or areas in which the fremitus is abnormally marked; the detection of such indicates the presence underneath of ***consolidation*** of spongy or vesicular lung tissue usually.

Consolidation of the lung tissue as a cause of marked vocal fre tus is best exemplified in *lobar pneumonitis* (pneumonia); in p *monary tuberculosis* it is commonly perceived over the solidified are In instances of the former the fremitus is usually felt posteriorly bel the scapula, and over a larger area than the latter, which also occ most frequently at the apices of the lungs, and anteriorly. In r cases of so-called *massive pneumonia,* in which the bronchial tu are plugged with exudate, the voice is feebly, or not at all, tra mitted through the solidified lung. In the later stages of tuberculo if the pulmonary *cavities* are large, superficial, and dense-walled, tactile fremitus may be increased (good conduction and consona by reflection of the vibrations from the walls). Increased fremi is also felt above a pleuritic effusion, the lung being compres against the thoracic wall, particularly in the infraclavicular regio over fibroid thickening, hemorrhagic infarction (pulmonary apoplex and edema of the lung (moderately); over solid tumors lying contact with and between a large bronchus and the chest wall, or, times, even a large band of pleuritic adhesion stretched between lung and the costal pleura; lastly, in some cases of chronic bronch with marked dilation of the bronchial tubes and adjacent consoli tion, these conditions are indicated by the increased fremitus.

Diminished Vocal Fremitus.—In the majority of instances, abn mally weak pectoral fremitus points to the intervention of someth between the lung and chest wall that interferes with the conduct of the voice vibrations to the surface. Thus, a thickened pleura, thin layers of an acute or subacute pleuritis, so diminish the fremit Again, since the bronchial tubes are the most important direct mea of voice transmission, diminution of their caliber, as from bronchi asthma, stenosis, or tumor compression may lessen the sign. Cavi in the lung that have become partly filled with liquid exudate ma fest a change from increased to diminished fremitus.

As the degree of fremitus over vesicular lung tissue depends part upon its tension, we sometimes find it impaired in marked e physema, owing to the loss of elasticity, in spite of the fact that alveoli are enlarged. But here the partial occlusion of many of bronchioles in this disease also operates simultaneously to prod the same result. In so-called compensatory emphysema or temporar exaggerated lung action, the tension being increased and the wh respiratory tract open and distended on that side, the fremitus increased at first, and, as recovery of the diseased side removes the reason for its continuance, later *diminishes to normal.*

It should not be forgotten that diminished vocal fremitus is not pathologically significant unless the voice is strong enough and the thoracic walls thin enough; feeble voice and dense, fat-laden walls are in themselves sufficient causes for weak fremitus, and then this is evident on both sides, although not unusually bilateral in emphysema, also.

Absent Vocal Fremitus.—Bronchial, pleural, and mural conditions may cause total abolition of fremitus.

In *occlusion of a bronchus* there is total absence of the fremitus over the area supplied by the occluded bronchus, as the oscillations of the voice are then entirely checked from entering that portion of the lung. The principal causes of closure of the bronchial tubes are the pressure of a large aneurism, mediastinal tumor, or enlarged glands; fibrinous bronchitis, blocking accumulations of mucus, and foreign bodies within the lumen.

Pleural effusions are the most frequent causes of absent vocal fremitus. Whether liquid or air, their presence in the pleural cavity interferes with the conduction of the vibrations to the chest wall, the denser medium of the latter inhibits their already weakened intensity, and the partially or completely collapsed lung, having lost its elastic tension, also fails to vibrate. Ordinarily, the encroaching fluid is either serofibrinous or purulent from inflammatory or tuberculous exudation; it may be a dropsical transudation (hydrothorax), or it may be partly (serosanguinolent) or wholly blood (hemothorax) or air (pneumothorax). A greatly thickened pleura will also suppress palpable voice transmission.

The palpation of the vocal fremitus is of the most practical value in the differential diagnosis of pleuritis and pneumonitis, diminution of the thrill or its absence, especially in the lower and posterior part of the thorax, being a cogent indication of the presence of the former affection.

It is needless to more than mention the deadening effect upon the tactile fremitus that various thickenings of the chest wall have, such as tumors, abscess, edema, and the like.

Pleural or Friction Fremitus.—Normally, the costal and pulmonary layers of the pleuræ glide smoothly over each other, so that no frictional vibration is perceptible to the hand. When, however, the pleural surfaces become inflamed, and roughened and thickened by a sticky or buttery fibrinous exudate, the friction produced by the respiratory rub gives rise to a palpable sensation to the examiner's hand. This variously described—often according to the intensity of the inflamm tion or of the respiratory energy—as of the lightest grazing or scratc ing or scraping character, or it may be of a dull, grating, or creaki character, like the bending of new leather. In some cases of t grating fremitus, the quick succession of jerky, detached sensatio is due to the fact that "even in circumscribed areas the roughen pleuræ do not touch each other at all points at the same time"; others, it is due to the irregular roughnesses of plates of calcareo infiltration in the pleura of very old chronic thickening and adhesio

The friction fremitus is best felt (and heard) in the axilla regions, about the fifth and sixth intercostal spaces, but also a lit to the front and below the scapular angle. It may have a to-and-f quality, synchronous with the respiratory rhythm, or it may be f only with inspiration, near the end. Deep inspiration usually creases the rub, although after a number of such inspirations t grazing surfaces may become smoothed out and the fremitus d appear; firm pressure with the hand apposing the inflamed pleu layers more closely, may also temporarily exaggerate the sensati Not infrequently the patient feels the friction as well as the p caused by it, and is able to indicate the exact seat of the trouble.

The disappearance of pleural fremitus, besides reduction by res ratory smoothing action and the absorption of recovery, may be to the separating effect of a progressing liquid effusion—the dry plastic pleuritis becoming serofibrinous—or to the formation of ad sions between the parietal and visceral pleuræ.

Bronchial or Rhonchal Fremitus.—This is a coarse or fine rattli sensation which may be felt all over the chest, especially in childr in diffuse catarrhal bronchitis. The vibrations are synchronous w the movements of respiration, although usually more distinct w inspiration, except in asthmatic cases. They are produced by vis mucus or abundant liquid exudation partially plugging or filli congested or inflamed bronchial tubes, disturbed by the ingoing a outgoing currents of air. As this agitation of bronchial secret gives rise to the audible râles (see under Auscultation) or rhonch variously pitched snoring, whistling, or bubbling sounds—when p pated the resulting vibrations are often designated as *rhonchal fre tus*. It is better elicited by getting the patient to take several d inspirations, and is more generally perceptible over the whole ch in children than in adults. In the latter, bronchial fremitus is usua due to a rather wide-spread chronic bronchitis associated with asth or emphysema, and not rarely with dilation of one or more portions of the larger bronchi (*bronchiectasis*), the discovery of which may be facilitated by noticing a more decided fremitus over that region because of the more sonorous gurgling in such a formation. This leads to the related subject of:

Cavernous fremitus, which is simply that felt over superficial cavities of the lung when near the apex, and in those whose chests are thin-walled and emaciated. The vibrations are distinctly localized, fine, and may disappear after a spell of coughing.

Bronchial fremitus may be differentiated from pleural fremitus by the following points: (*a*) *Location.*—The former is more diffused, the latter unilateral; and when the fremitus is circumscribed, its occurrence over the upper and anterior portions of the lung indicates a bronchial origin, the pleural being lower, and either axillary or posterior. (*b*) *Character.*—Bronchial fremitus is more continuous, pleural more jerky and interrupted. (*c*) *Rhythm.*—Bronchial is felt almost as constantly during inspiration as during expiration, while the pleural is more apparent at the end of inspiration or the beginning of expiration. (*d*) *Behavior in Coughing.*—Cough accompanied with free spitting diminishes bronchial palpable râles; without such looseness they become weaker at some points and present or exaggerated at others, on account of displacement of the mucus; pleural fremitus is not so modified by cough.

Tussive Fremitus.—This means the palpable vibrations transmitted to the chest wall by the act of coughing in itself. It is of very little clinical importance except in cases of aphonia, when it may serve to indicate physical changes otherwise determinable by the vocal fremitus, increase, diminution, and absence of the latter constituting a superior means of ascertaining the changes similarly affecting the tussive fremitus under usual conditions.

Succussion, or Splashing Fremitus.—When sensation of a splashing wave against the thoracic wall is felt with the shaking of the body, it signifies the presence of air and liquid in the pleural sac (*hydropneumothorax*; *pyopneumothorax*).

Fluctuation.—*Simple fluctuation,* as elicited by surgeons in the detection of superficial abscesses and cysts by an alternating method of finger pressures, may be perceived in cases of pleural effusion where the intercostal spaces bulge, as from an empyema. The so-called "*peripheric fluctuation*" (Tarral) is elicited by "giving a quick, sharp fillip in an intercostal space, perpendicular to the surface, when a sensation of fluctuation will be transmitted to a finger of the other hand firmly applied to the surface in the same space, a short distance from the point percussed." While this may dete mine the presence of a tense pleural effusion, it is particularly applic ble in the detection of a hydropneumothorax. A thickened pleu would interfere with the manifestation of this sign.

Resistance.—The presence of pulmonary or pleural tumors m sometimes be localized by an increased sense of resistance over t growths, especially where the ribs and interspaces may be bulge their hard or soft consistence may indicate the carcinomatous sarcomatous characteristics.

Likewise, occasionally the diagnosis of a pneumonic consolidati may be aided by a relative increase of resistance to the palpati finger, elicited over the corresponding interspaces, provided the lu is closely apposed to the chest wall. The tension of a large pleur effusion is still more evident.

Again, a limited area of pitting with finger pressure slowly an firmly applied, with marking of the cutaneous furrows, and a co sistence not unlike that of dough or putty, usually points to a pleur collection of pus. This occurs at the base of the chest, and is accom panied with slight swelling and inflammatory redness and tendernes

Pulsation.—Palpation confirms the ocular detection of pulsatio abnormally located. Thus is recognized the *pulsating empyema.* T heart being displaced to the right, in certain rare cases marked puls tion has been felt at the sternal portion of the infraclavicular an mammary regions; sometimes pulsations are found even on the le lower portion of the thorax, and here are less likely to be mistake for aneurismal pulsations. A pulsatile movement of the lung h been noticed in some rare instances of pneumonitis and intratho cancer.

CHAPTER IV

PERCUSSION

Fundamental Principle.—It is that in common daily life we learn to distinguish many objects by the different sounds they emit when struck; such physical conditions as their material quality, density, thickness, partial or complete hollowness, are so recognized. This principle is applied to the physical diagnosis of the human body; organs are located, their size, shape, and outline mapped out or carefully inferred, and such properties as mentioned before determined in their normal and abnormal relations and variations and significances.

The fact that with this method of examination began in reality modern scientific diagnosis, deserves a brief look backward, as an introduction to present procedure.

Historical Note.—We owe to the Viennese physician, Auenbrugger, the discovery of the method of percussion, as described in his memorable work entitled *Inventum novum ex percussione thoracis humani*, etc., which appeared in 1761. The importance and practical value of the method, being disputed and ridiculed into neglect, failed of any recognition for nearly fifty years, when Corvisart, body-physician to Napoleon I, in 1808 gave it substantial impetus by reviving and commending its use and improving its application. His French translation soon led Piorry to develop further the method into the *topographical percussion*, and, in 1828, to the invention of the *pleximeter*. Since then, through the wider observations and deeper understandings of such men as Skoda, Wintrich, Traube, Gerhardt, Weil, Walshe, Sansom, and Flint, chest percussion has been elaborated into a procedure as precise as auscultation, very often and usually quite as delicate and difficult to practise. Most important advances in the percutory practise and interpretation were initiated by Piorry and Skoda; the introduction and development of mediate palpatory percussion (see p. 84) in the examination of the abdominal as well as the thoracic organs being especially noteworthy of the former; the latter's discovery of the physical causes of many of the qualities of percussion sounds and their normal and pathologic connections, and his exhaustive contributions to the subject of tympanicity (1839) were of distinguished importance.

Definition.—*Percussion* is the practise of striking or tapping the body to elicit vibrations, to obtain information as to the physical condition of the underlying organs. It probably requires more perseverance in acquiring manipulative skill than any other method of physical examination. Such facility is requisite not only to bring out the true and characteristic sounds, but also to allow the attention of listening to be concentrated upon those sounds which it is the purpose of the method to study. The mind of the examiner must be freely receptive and intelligently alert, and not diverted by the conscious and zealous energy of awkward and laborious manual effort. To this end the student needs incessant practise, assiduously at the outset, so that his technic becomes a matter of automatism, almost, along the lines given in the details of method to follow.

METHODS OF PERCUSSION

There are two methods of percussion, *immediate* or *direct*, and *mediate* or *indirect*.

Immediate percussion, by which striking of the chest is performed directly with the tips of the fingers (usually the second finger), or even with the flat of the hands, was the method employed by Auenbrugger and his early followers. It is seldom used now, not only because of the annoyance to the patient, but because of the more agreeable and, so far as results are concerned, greatly superior mediate method invented by Piorry. Nevertheless, there are circumstances in which this method may be resorted to with advantage. Thus, where there are extensive and marked differences between the two sides of the chest, a rapid tapping or lightly slapping with the palmar surfaces of the hands will often elicit quite satisfactorily the distinctive sounds, as of the presence of a pleuritic effusion. The clearer, more resonant tone over the upper part of the chest, shading abruptly into the duller note at the edge of and over the cardiac and hepatic regions, enables one to delimit the lung-heart and lung-liver boundaries in thin individuals fairly well. Furthermore, the fact that the sounds thus produced are virtually vibrations communicated by the bones of the thorax, points to the added value occasionally of directly tapping the clavicles, ribs, sternum, and scapulæ; lightly percussing the clavicles not infrequently indicates the presence of subjacent tubercular infiltration, as I have demonstrated; enlargements of the heart, aortic aneurism, large effusions, and mediastinal tumors may also encroach under the sternum so as to be inferable from the results of immediate percussion. The direct percussion of the softer parts, as the intercostal spaces, does not yield so clear and recognizable tones.

Mediate percussion may be practised in three different ways, as follows: (1) Finger percussion; (2) finger-pleximeter percussion; (3) hammer-pleximeter percussion. That is, the interposed medium —the thing applied on the chest to receive the stroke—may be either a finger or a *pleximeter* made of some solid material, conveniently shaped for the purpose; and the striker, or *plexor,* may be either a finger or special hammer. Each of the three modes may yield satisfactory results if applied with respective skill and care, and when such proficiency is acquired it is wise to stick to the particular mode throughout one's work, although it may be said that facility in the use of the simple finger method of percussion makes it much easier to apply the others than *vice versa.* To change methods and instruments frequently tends to mediocrity in the use of all of them.

The "clinical supremacy" of the fingers as plexor and pleximeter lies in the fact that they are, in the first place, naturally the most handy; instruments artificially made are apt to be inconveniently misplaced, lost, or forgotten. Secondly, fingers, with their sense-organs in close contact with the region percussed, afford more delicate and intelligent results than lifeless appliances; especially valuable in this connection is the appreciation of the degree of the sense of resistance while percussing, to be referred to more fully later. Thirdly, the pleximeter finger is adapted readily to accurate fitting on the thoracic surface, excepting at times the supraclavicular fossa. Finally, the homogeneity of flesh and bone against like structure of the thorax avoids the confusion of the percussion note of the underlying organ with the clacking sound obtained by striking a hard-rubber pleximeter, for example.

Pleximeters consist of small plates or upright pieces of gutta-percha, ivory, glass, or wood, round, oblong, or elliptical in shape, so as to be applied to the intercostal spaces. The pleximeter invented by Piorry was a thin, circular, or oval plate of ivory about 2 or 2½ cm. wide, fitted at opposite points with two vertical ears roughened on their outer aspects, and just large enough to permit the thumb and finger to hold it steadily and apply it evenly and firmly to the surface. Sansom's pleximeter, a combination of hard-rubber pillar, and plates of unequal size, is very useful in percussing over small depressions, as above the clavicles, the only region where finger percussion may sometimes be difficult. For the same purpose the double pleximeter of Seitz, like a small tongue spatula with a double curve, will occasionally be found convenient. As helpful as any is a cylindrical piece of india-rubber, about the diameter and two-thirds the length of a little finger, pierced lengthwise with strong wire, the ends of which are twisted together to form a handle, after extending a short vertical distance above the rubber, and then bent at right angles.

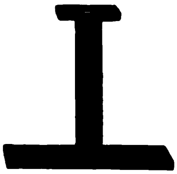


Fig. 18. — Sansom's Pleximeter. (Tyson.)

The percussion hammer was invented by Wintrich in 1841. It is generally of moderate weight, has a firm but light handle of wood or aluminium fixed to a heavier metal head tipped with rubber, or ending in a ring containing a cylindrical piece of india-rubber.

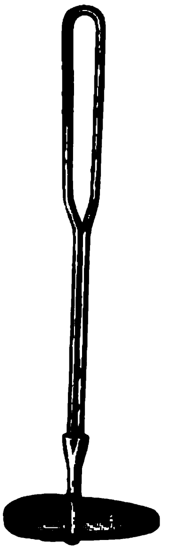


Fig. 19.—Wintrich's Percussion Hammer. (Tyson.)

Technic of Mediate Percussion.—(1) Finger Percussion.—*The position* of the *patient* should be a sitting or standing one whenever possible, with the chest bare, or, from motives of delicacy, a thin undershirt may be permitted, but it must be soft and smoothed out evenly. Again, should there be any danger of chill, a part of the chest may be exposed at one time, provided the corresponding region on the opposite side is exposed at the same time, so as to facilitate comparative percussion, or, while the anterior surface is being percussed, for example, a small blanket, sack, or shawl may protect the back and shoulders. Patients who are too sick or weak to be examined in the sitting posture should be placed perfectly level, or semirecumbent, the body and limbs straight and the shoulders quite even. It is an important fact to be noted that the sounds are apt to be dulled in percussing patients who are in sunken contact with heavy bedding and pillows.

Whether standing, sitting, or lying, the patient should have a comfortable, easy, relaxed position. If sitting, and even standing sometimes, it is necessary to use a back support, otherwise the firm pressure of the examiner's pleximeter finger may cause confusion of results by the patient's swaying the body or unsymmetrically holding tense the chest muscles for bracing.

In percussing the *front* of the chest, the patient standing or sitting, it is often particularly necessary to obtain muscular relaxation because of the habit some persons have under examination of rigidly swelling their chests out as if ashamed to present less fulness. The body and head must be erect, without turn or twist of either, the face as well as the chest pointing forward; the shoulders not the least shrugged or held back, but allowed to fall easily and symmetrically forward, while the arms hang loosely at the sides.

To percuss the *lateral* and *axillary* regions it is necessary to have the arms drawn back, or still better, raised by the hands clasped over the head.

In percussing the *back*, the patient should stoop forward with arms folded and a slight shrug of the shoulders, thus exposing more of the chest by the outward lift of the scapulæ.

The effects of the variation of attitude upon the movable borders of the lungs, and their relations to such movable adjacent organs as the heart and liver, will be mentioned later (see *topographical percussion*).

The *pleximeter finger* is usually the middle one of either hand, according as one is right- or left-handed. It should be placed horizontally between the ribs, firmly and flatly, and with the same degree of pressure and on the same parallel at the same relative position to any given vertical line on the two sides before a higher or lower interspace is percussed, and thus always *comparing similar parts on the two sides.* By percussing in this manner only can the purest and most distinctive lung sounds be elicited, so far as the pleximeter finger is concerned. On thin chests it is practically impossible to apply this finger evenly except parallel to the ribs, whether between them or on them. The true note is modified by the cushion of air under a finger placed slantwise or vertically; also when the bones are percussed; but if the comparative method is strictly adhered to there is less likelihood of inaccuracy. Not infrequently, however, it is convenient or advantageous to apply the finger at various angles to the ribs, as in outlining organs; in such cases the tip of the finger may be laid in the interspace so as to avoid pressure against a rib; in stout individuals, where the chest wall is thick and smooth with flesh, this is not so necessary.

In some parts, as the supraclavicular and axillary, to avoid a constrained difficulty of percussion, the side of the pleximeter finger may be laid on the tissues, and the stroke received upon the lateral bony prominence of the terminal phalangeal joint.

Valuable information may more readily be obtained sometimes by using the four fingers of the pleximeter hand laid in as many interspaces, each finger being percussed successively with a single stroke. Slight differences in sound near the borders of the lungs and adjacent are thus especially well brought.

The *plexor finger* is usually the second or middle one of the right hand (for right-handed examiners). Beginners and those who have never acquired that facile resiliency of percussion stroke so essential for skill and satisfactory results, are prone to use two or three fingers to percuss with, endeavoring to make heaviness of blow bring out the sound; whereas, if from the first close and patient attention to the details of a proper technic be given, and the diligent practise of them be persevered in, a *clearer note* from a lighter, single-fingered stroke may be elicited, and the patient's discomfort, and resentment perhaps, avoided. Habitually does one see the sophomore "med," when commencing to learn percussion, imitate, as he thinks, his teacher's method after a superficial observation, and, without a concentrated study of the conditions and factors of successful percussion, proceed vigorously to practise in a faulty and discouraging manner that daily becomes more and more difficult to correct and eradicate.

What are those points to be shunned that are at the same time so easily drifted into? In the first place, a constrained, awkward, and unsymmetrical position on the part of the examiner with regard to the patient. The former should so place himself that perfect freedom, fulness, and comfort of action may be obtained, and maintain as nearly as possible equal distances between the listening ear and the respective parts percussed.

(*a*) The plexor finger may not be flexed enough; it should be bent sufficiently at the second or middle joint so that the tip of the finger falls perpendicularly upon the pleximeter finger, the distal joint being fixed as rigidly as possible; any variation from this practise only weakens and alters the true character of the sound, a slanting blow being a flabby one.

(*b*) Too often the stroke is heavily delivered from the elbow, or shoulder even, which, with the preceding fault, causes the patient to be disagreeably pushed instead of percussed; the movement should *spring from the wrist* and be simultaneously reenforced by movement at the knuckle. It is only by accomplished wrist action that uniformity and flexibility of technic are acquired, and reliability and nicety of results obtained.

(*c*) A lingering, poking, slow stroke—that is, slow in lifting from the pleximeter finger after the delivery—is an essential fault; the blow must be quick—that is, short in duration of contact; therefore it must have the rebounding, elastic quality, as of a piano hammer without the dampening effect of the soft pedal; or the tap should be given with such immediate removal of pressure after contact, as if one were percussing resilient rubber—smartly.

(*d*) Bearing in mind that we must compare similar parts on the right and left sides of the chest, *prolonged percussion over one spot may prolong the impression to the ear so as to interfere with its sensitiveness to adjacent differences*: the location of the fingers should be changed after two to four successive strokes; indeed, it is better to make frequent brief comparisons back and forth than to be satisfied with one or two prolonged hammerings.

(*e*) The percussion blows should be delivered with moderate rapidity—about two per second. When the blows are made too rapidly interference waves hinder the production of clear, distinct sounds.

A few *further considerations* require careful notice.

The *force* of the strokes ordinarily should be moderate, always uniform on the regions compared, never painful to the patient. Exaggerated force must be particularly guarded against when two or three plexor fingers are used at once, and then care should be exercised to strike with them evenly. For simple clinical purposes the light, practised stroke given with one finger is forcible enough; for purposes of demonstration to a small group of listeners, even, I have found it sufficient.

Whether *forcible* or *gentle percussion* is to be employed depends upon the special conditions, and the object in view in each particular case. In a general way the following considerations may serve as guiding rules, reserving other more specific ones for the subject of topographical percussion of the lungs. The stroke should be rather *forcible* in examining those regions of the chest that are thickly overlaid with flesh—the decidedly muscular and the decidedly fat individuals. The clear, resonant pulmonary sound cannot be well elicited, for instance, in the mammary and scapular regions for obvious reasons, unless forcible percussion is used to make the vibrations penetrate the thick wall of the chest. On the other hand, lighter strokes suffice in the infraclavicular and axillary spaces. The percussion blow must also be much less forcible in children, in whom the thoracic walls are relatively thin and elastic, than in adults, except those who are very emaciated. Indeed, energetic strokes in such cases set into vibration such a large volume of surrounding tissues that the quality and underlying physical causation of the sounds to be defined over a small, circumscribed area are hidden or confused.

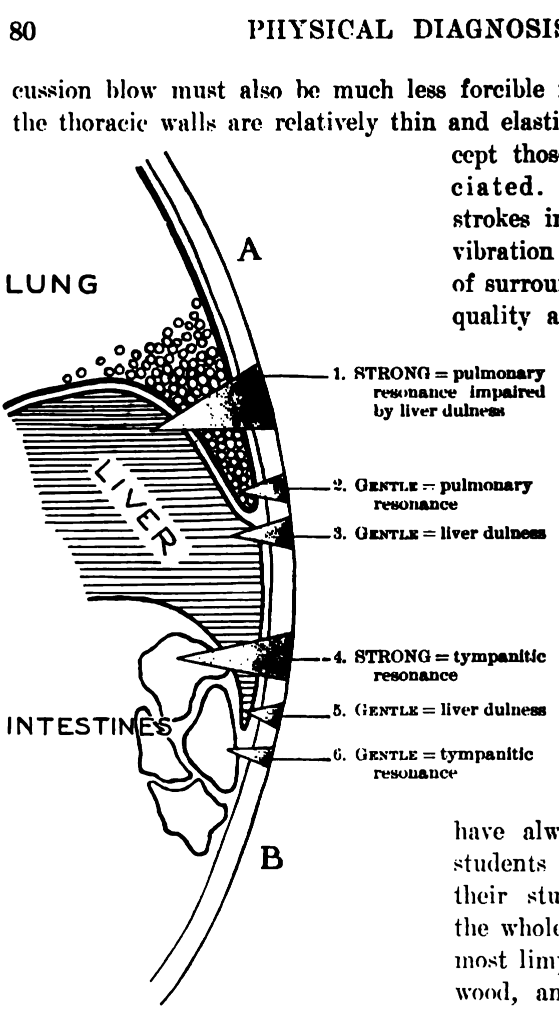

A
LUNG
LIVER
1. STRONG = pulmonary resonance impaired by liver dulness
2. GENTLE = pulmonary resonance
3. GENTLE = liver dulness
4. STRONG = tympanitic resonance
INTESTINES
5. GENTLE = liver dulness
6. GENTLE = tympanitic resonance
B

FIG. 20.—DIAGRAM SHOWING THE RATIONALE AND UTILITY OF VARYING THE FORCE OF THE PERCUSSION STROKE. A strong stroke as at 1 develops deep dulness and locates the upper border of the liver, while a gentle stroke in the same spot gives only pulmonary resonance. A gentle stroke at 2 gives pulmonary resonance, and the next gentle stroke, 3, gives liver dulness, thus locating the lower edge of the lung, while a powerful stroke at 2 will give mainly liver dulness. Gentle stroke 5 is slightly dull, and gentle stroke 6 is unmistakably tympanitic, thus marking the boundary-line between thin edge of liver and air—containing intestine, while strong stroke 4 elicits tympanitic resonance. (Butler.)

Nothing short of ease and elasticity of wrist percussion will enable one so to vary the force of the stroke that under all circumstances satisfactory results may be obtained. For this purpose I have always recommended to students constant practise at their study tables by laying the whole forearm flat and almost limp upon the uncovered wood, and thus perseveringly getting the automatic wrist and finger habit. Variations of the blow may be made both in force and location, as over a table leg, to note the different sound effects; also with a thin table cover applied, and, raising the arm, striking a book or a pleximeter.

A contraindication to percussion should be mentioned here, namely, in those cases of pulmonary tuberculosis who shortly before examination have suffered from a hemorrhage, or who may at the time be bleeding from the lungs. Especial care required in certain cases of pneumonitis, cavities, etc., will be referred to further in connection with those subjects.

(2) Finger-Pleximeter Percussion.—When, as intimated before, it becomes awkward, difficult, and unsatisfactory to apply a finger as pleximeter; or when, as sometimes happens, the interposed finger becomes painful, swollen, and even ecchymotic, as I have seen occur in dispensary and ward hospital work, where numbers of chests have to be percussed, the use of an artificial pleximeter becomes a necessity. The part of the finger usually struck is just back of the nail, where normally there is a rich capillary circulation; and therefore prolonged percussion here may easily set up a painful hyperemia during a series of examinations, although this can be prevented by striking the middle phalangeal bone instead of the distal one, habitually or temporarily. However, it is in such cases that a Sansom's pleximeter, for example, may be substituted.

In applying the pleximeter, the same care regarding firmness and evenness should be observed as with the finger. It should not be forgotten that because of its hardness an ivory or celluloid or gutta-percha pleximeter may be pressed so firmly as to hurt; and that because of its coldness, according to the weather, it may produce disagreeable chilliness of the surface, and must be hand-warmed before using.

Again, judicious allowance must be made for the sound produced in striking the pleximeter itself, and not let its clack overshadow the chest sounds from within. This may be guarded against, in one way at least, by preserving shortness of the nails, and by insuring the contact of the plexor finger at its rounded end, about midway between the nail and the palmar pulp.

(3) Hammer-pleximeter Percussion.—This method is the easiest to master, but is generally too ostentatious, and may be terrifying to timid patients. Nevertheless, it has a few occasional advantages. Thus, until finger percussion has been learned with skill sufficient to elicit the true sounds of a given region the student may practise the recognition and discrimination of those sounds brought out more distinctly with the percussion hammer and pleximeter—train the ear while the fingers are catching up. The hammer taps should be delivered with equal, gentle force, equal frequency and rapidity on the two sides and similar areas of the chest.

When for any reason, as for the detection of deeply seated growths, consolidations, and cavities, it is desired to make the vibrations penetrate deeper into the thorax, hammer-pleximeter percussion is a useful adjunct. In other cases, the erroneous results one is apt to be led into by the too penetrative power of this method setting into vibration too large volumes of resounding substance, and the disadvantage of the sense of resistance experienced in finger percussion, may be avoided by percussing with a very light stroke, and by grasping the hammer not at the end, but at the middle of the handle, and at the same time keeping the index finger closely applied to the hammer head.

We may *summarize* by stating that finger percussion is the most valuable and generally useful method, really must be acquired sooner or later, and although the most difficult to gain technical skill in, when once achieved yields the best results, and makes easy the practise of other methods, which is not true conversely. Again, the importance of always *comparing* corresponding portions on the two sides under the same conditions of posture and technic must not be forgotten.

Respiratory Percussion.—Ordinary quiet respiration does not affect the constancy of the percussion sound, which is of average quality in this respect. But to make assurance doubly sure, the two sides of the chest should be percussed comparatively during the same stages of the breathing act; that is, a note elicited during inspiration on the right side should be compared with one during inspiration, and not expiration, on the left side, and *vice versa*, especially if the breathing is a little exaggerated.

The term *respiratory percussion* is more specifically applied, however, to the perception of differences of sound at the end of a deep inspiration or of a full expiration, the patient holding the breath in each instance. The conditions affecting this test in general will be noted in discussing the attributes of sounds obtained in percussing over the lungs.

Auscultatory or Stethoscopic Percussion.—This useful method, which deserves much more practical attention and application than it has received, was introduced by Drs. Cammann and Clark, of New York, in 1840.

The method combines listening with the stethoscope with percussion. The original stethoscope of Cammann was a solid piece of wood about six inches in length and three-fourths of an inch in diameter, with an ear piece firmly attached to it. The binaural stethoscope is now used. The technic consists in placing the chest piece of the latter (the bell of which should be small enough to rest snugly between the ribs) over the organ whose outlines are to be studied, and percussing lightly toward it from different points beyond the supposed borders of the organ, tumor, or other pathologic formation. At times it may be more satisfactory to have an assistant hold the chest end of the stethoscope; or, as some prefer, so as to avoid the modifying vibrations of the bony thorax, the percussion notes may be listened to with the bell of the stethoscope suspended a little above and near the point of percussion. Again, as with the use of the phonendoscope (see instruments for auscultation) for the same purpose, instead of pleximetric percussion a stroking or scratching with the index finger may be done upon the skin near the chest piece, the changes of sound conducted being thus noted.

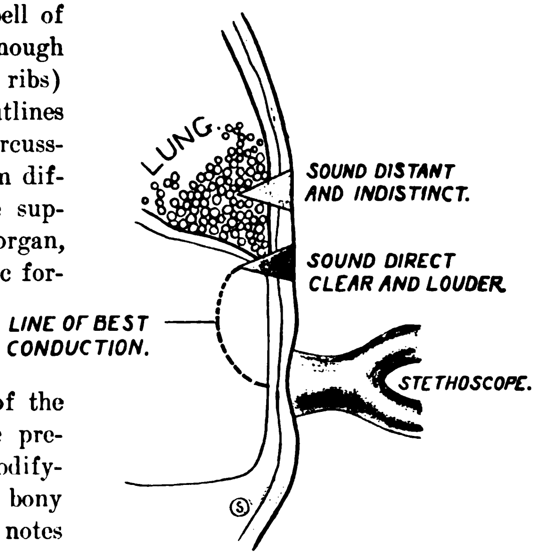


FIG. 21.—DIAGRAM SHOWING THE THEORY OF AUSCULTATORY PERCUSSION. The organ over which the stethoscope is placed may be either solid or hollow. (Butler.)

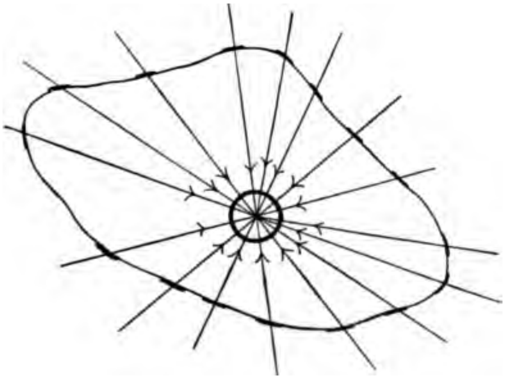


FIG. 22.—DIAGRAM SHOWING THE LINES TO WHICH AUSCULTATORY PERCUSSION SHOULD BE CARRIED IN ORDER TO OUTLINE AN ORGAN. The central circle represents the chest piece of the stethoscope. (Butler.)

By this method the sounds produced are louder and more distinct than when communicated simply through the air without the aid of a stethoscope. Besides, *changes* in *quality* and *pitch* are noted more readily in passing the boundaries of

organs, and hence the latter may be marked more precisely. Th
character and intensity of the percussion note continue the same s
long as the stethoscope and finger are over the same organ or patho
logic formation; but become altered and weakened at once when th
limits are reached by either. The value of the application of thi
delicate technic in the outlining of such organs and morbid condi
tions as the heart, stomach, liver, and spleen, and thoracic aneuris
is quite evident, and as employed there will be described under thei
topographic examination. However, in delimiting the lung border
and even the lobes of the lungs, in discriminating a pulmonary con
solidation from a pleural effusion, and both from normal or enlarge
liver, auscultatory percussion may be of marked service. (See Fig
21 and 22.) As Cabot points out, to insure reliable results from thi
method the percussion must be made along arcs of circles in approach
ing the presumed borders of an organ, thus keeping the chest piec
at all times and in changing locations at an equidistant center, other
wise the intensity and quality of the sounds transmitted will vary wit
the longer or shorter lines of distance and so give the false impres
sion of different organic tissues.

Palpatory Percussion.—Not only do we study the sounds elicite
by percussion, but the *sensation of resistance* to both fingers, esp
cially the plexor finger, is also appreciated. It partakes of palpatio
as well as of percussion. It is influenced by the *degree of capacity*
the underlying parts to vibrate; the sense of resistance is slight ove
air-containing, resilient tissue, while the denser the structure th
greater the resistance.

It is the estimation of this amount of resistance which enters mo
essentially into the performance of *palpatory percussion*, which r
sorts to a sort of pushing stroke. Two or three fingers of the plex
hand are partly flexed so that the pulps strike or push against th
pleximeter finger from a shorter distance above the latter than i
ordinary percussion, and the rebounding stroke is also replaced b
longer contact—"palpating stroke." To some clinicians palpator
percussion is often more delicate than auditory percussion for th
purpose of mapping out organs. The light, inaudible tapping elici
vibratory sensations, the perception of which is attributed by the Ru
sian, Tranoff, to a sort of dermal hearing—the ability of the ski
to differentiate the quality, force, and pitch of peripheral vibration
When forcible auditory percussion is contraindicated, as in hemo
tysis, or when it is useless, as in crying children, or when the phys
cian's sense of hearing is defective, this method may be invaluabl

though every physician should endeavor to cultivate his "skin-sense of vibrations." The method is not as commonly used in this country as in Germany.

Superficial and Deep Percussion.—These terms are used frequently to describe respectively the lighter blows given to elicit sounds only from tissues directly under the chest wall, as the thinner layers of lung partly covering the heart and liver, and the heavier blows given to bring out the sounds of pathologic formations deeply situated in the lungs, or of solid organs overlapped considerably by the lungs.

THORACIC PERCUSSION SOUNDS

The foundation fact upon which the practise of percussion of the body depends is, that some of the principal organs contain air or gas, or both, and that there are more or less characteristic differences in the sounds produced by percussing over them on account of certain normal (mainly anatomic) conditions, and pathologic changes affecting them or contiguous organs or tissues.

To understand and interpret these variations of sound one must be acquainted with the physical properties that characterize them, for it is upon a clear perception and analysis of these that their diagnostic significance rests. After a time they become distinguishable with a speed that seems intuitive, but is simply the outcome of an established and assured habit of painstaking and precise recognition.

Percussion over *air-containing* structures gives rise to a more or less *clear* tone, of a resounding character over normal lung, drumlike over the stomach and bowels.

But when we percuss over *solid, airless* tissues or organs, the sound elicited is *absolutely dull* or *deadened,* like that obtained by striking upon the thigh, hence also "thigh sound." It lacks the uniformity and regularity of vibrations of the clear, almost musical tones from percussed organs containing air, as the pulmonary vesicles; it is a noise, dead and flat.

As there are many gradations between the tonelessness elicited over solid organs and the clearness of normal pulmonary resonance, and as both extremes, and the intervening grades of "impaired resonance" or "relative dulness," are really recognized by their *physical attributes,* an analytic study of these is necessary before their special, practical application is made in the percussion of the normal and pathologic lungs. However, the relation of these acoustic elements to illustrative healthy and morbid conditions of the lungs will be designated in a general way, in preparation for the easier apprehension of their significance in the diagnosis of the special pulmonary diseases.

ATTRIBUTES OF PERCUSSION SOUNDS

In judging percussion sounds, we note four attributes: (A) *Quality*; (B) *intensity* or *volume*; (C) *pitch*; (D) *duration*. None of these can actually or adequately be learned from descriptions alone: they must be recognized by ear practise, to which the definitions and illustrations here given may render intelligent direction and perception.

(A) **Quality,** in physics, is that peculiar, essential property which distinguishes sounds from different sources, although the other properties may be alike. It corresponds to the *timbre* of a musical tone, by which we appreciate its instrumental source, whether of the stringed, brass, or reed class, whether a single instrument of each class, as a violin, trombone, or clarinet, regardless of similarities or alterations of pitch and intensity. Quality is that distinct characteristic of a sound which makes it what it is, and which it cannot lose "without ceasing to be."

Given the associated structural conditions of a pipe-organ or harp, for example, the tones of which one has heard often enough to be familiar with, and as either instrument may then be readily recognized by its tonal quality, though not visible to the listener, so one must strive to gain familiarity with normal and abnormal physical conditions and changes of the lungs by and through the quick perception of, and swift deduction from, the attribute of quality.

As indicated before, there are two extremes of quality heard in percussing over the lungs (or body): (1) the *clear* sound of air-containing tissue; (2) the *dead* or *absolutely dull* sound of airless tissue. The term clearness is, of course, a relative one, its degree depending upon the volume of air-containing tissue, other things being equal.

We meet with two distinct qualities of clear sound: (1) *nontympanitic* or *resonant* sound; (2) *tympanitic* sound.

(1) Resonant Sound.—The former of these, more commonly designated as the *normal pulmonary resonance*, or *normal vesicular resonance*, is the characteristic clear lung sound elicited by percussing over healthy vesicular air structure physiologically distended. Although its quality has been likened to the sound obtained by gently

striking a drum covered with a thick woolen cloth (Auenbrugger), or to that produced by percussing over the upper crust of a loaf of bread covered with a towel (Flint), nevertheless its *sui generis* quality must be heard and learned by experience from each individual examined rather than from any description, however discriminating. Pulmonary resonance is distinctive enough to permit variations from the normal to be easily recognized.

The quality of the normal pulmonary resonance is probably influenced by the peculiar sound contributed by the bony formation of the chest wall, as when the slightly inflated lung that has been removed from the dead body is percussed, the sound is drumlike or tympanitic.

The clearness, though not the distinctiveness of note, is modified a little also according to the locality percussed, owing to the effect of intervening thoracic bone, muscle, and fat. This will be pointed out more appropriately under topographic percussion. The typical quality of normal vesicular resonance is usually best heard in percussing over the left infraclavicular region, or below the scapular angles in chests that are not fat or too muscular. Sometimes I have found it helpful in demonstrating the pulmonary resonance to percuss over the empty stomach, and thus compare the clear, tympanitic quality of the latter with the characteristic reverberating variety of the former; the differences of sound are pronounced, so that their pathologic association may more readily be detected, as in the occurrence of cavity of the lung (tympanitic quality with high pitch).

The *cause* of the normal vesicular resonance is probably the combined vibration of the chest wall, alveoli of the lungs, the bronchi, and their contained air. Therefore, the clearness or fulness of resonance will depend upon the conditions of these elements.

The *modifying conditions* which give rise to variations in the tone of resonance in health are the following four:

(*a*) *Thickness of the Chest Wall.*—The greater the thickness the duller the resonance, and *vice versa.* Muscularity, unless very marked, does not influence clearness very much. On the other hand, the thick layers of nonelastic adipose tissue in stout individuals dulls the resonant quality considerably. *Per contra*, percussion over the emaciated chest of a patient with healthy lungs, but dying with cancer in the abdomen, elicits very clear resonance with ready facility.

(*b*) *Influence and Resilience of the Osseous and Cartilaginous Framework.*—Since, as mentioned previously, the healthy lung removed from the body yields a tympanitic or drumlike sound when percussed, it is owing to the material influence of the thoracic wall in the living subject that percussion over its surface corresponding to the pulmonary regions elicits the special quality of the vesicular resonance, or "muffled" tympanitic sound, as it has been called. It should be borne in mind that bone, when thinly covered and percussed, has a peculiar resonance of its own. This is particularly marked as regards the sternum, and, to a slightly less degree, the clavicles and ribs. The close and cagelike framework of the bones of the thorax permits the vibrations of a percussion blow to be transmitted generally over other portions of the bony thorax. It is for this reason that the percussion strokes should be delivered mostly in the intercostal spaces so as to get the purest lung sound. The sternum acts as a natural sounding-board, and unless the anterior mediastinum is practically filled with a mass of firm tissue the sternal note is uniformly and peculiarly resonant, seemingly gathering up and intensifying the totality of pulmonary and mural vibrations set up.

The clearness of the lung resonance likewise depends upon the *resilience* of the osseous thorax, and this upon the age of the subject. In children, whose ribs are very flexible, the resonance is decidedly clearer than in adults; on account of the more rigid and resistant conditions of the chest wall in the latter, especially in the aged, there is more interference with the purity of the lung sound on percussion, and an overshadowing prominence of the "wooden" bony sound.

(*c*) *Amount of Air Within the Respiratory Tract.*—The vesicular resonance has a clearer quality in persons who are good breathers—whose respiratory expansion and vital capacity are good. We usually find this in athletes, and others whose occupation, healthful exercise, and physical culture compel them to fill their lungs well. The resonant lung quality is lacking, on the other hand, in those who are deficient in the volume of respiratory air: full lungs produce full vesicular resonance.

(*d*) *Presence of Adjacent Organs.*—That the characteristic pulmonary resonance is diminished in clearness near the borders of such solid organs as the heart and liver is an important consideration in estimating their respective limits. Again, the influence of the adjacent distended stomach upon the left lung sound is to superadd its drumlike tone, which thus develops a tympanitic resonance, principally over the lower half of the chest, anterolaterally.

(2) TYMPANITIC SOUND.—The *tympanitic sound* exists normally and typically over the stomach moderately distended with air or gas, and not over the lung except pathologically. With less fulness of quality, tympanitic resonance is also heard in percussing over the

colon and larynx, also over the distended cheeks, this variation of quality being due to variations of the attributes of intensity and pitch because of smaller volumes of air.

Tympanitic sound simulates very closely that of the tympanon or kettle-drum, and approximates a musical tone. Its clearness depends upon such factors as influence the pulmonary resonance, especially the thickness of the intervening tissue and the volume of contained air; besides these, the tension of the air- or gas-containing walls is important.

Two subvarieties of the tympanitic sound are noted, namely, the *open* and the *closed.*

The *open tympanitic* sound occurs in percussing over cavities containing air or gas, if surrounded by walls "moderately smooth and capable of reflexion, and if they communicate with the external air through an opening, the walls being stiff or yielding."

The *closed tympanitic* sound is produced over cavities or organs without communicating freely with the external air by openings, the walls being thin, membranous, and not too tense. Pathologically, lung cavities in direct communication with bronchi are examples of causes of the open tympanitic sound, while closed, isolated cavities and closed pneumothorax, as well as the normal state of the stomach, exemplify the second. These qualities are recognized mainly by differences in pitch, as will be pointed out in describing that attribute.

(3) THE DULL OR DEAD SOUND.—The *absolutely dull* or *dead sound* has the toneless quality and does not occur over normal lung, simply because such lung always contains more or less air; and yet this qualification implies that the degree of clearness of quality does vary according to the amount of air present, principally, in a given portion of the lungs; hence the commonly used term *relative dulness* for all intervening grades between clearness less than absolutely full and pronounced and dulness absolutely flat and dead, as obtained over solid, airless tissues or organs. The latter are exemplified in the case of the thigh sound obtained in percussing over the skeletal body coverings, over uncovered liver, heart, and spleen, and over the following pathological conditions: (*a*) The interposition of liquid or solid material, as pleuritic effusions or thickening, between the lung and chest wall, (*b*) or the infiltration of vesicular tissue and air-spaces with solid substance, as in the marked consolidation of pneumonitis.

While the terms dulness, flatness, and deadness are used interchangeably to indicate the absence of air, in common usage a dull sound is more particularly elicited where the resonance is very positively diminished; a dead or flat sound where we percuss over absolutely airless organs, solid growths, and liquid accumulations, the latter corresponding to condition (*a*) and the former to condition (*b*) of the preceding paragraph.

Clinical language also employs such qualifying words to express varying degrees of dulness as marked or considerable, moderate or slight, the two last terms meaning virtually relative dulness, that is, the presence of a slight amount of air directly beneath or adjacent to the part percussed, so that the sound possesses just sufficient resonance to escape being a noise—toneless.

In the case of very fat persons, owing to the great thickness of the chest walls, percussion over normal lungs with exaggerated force may yield only a deadened sound, the vibrations not penetrating deeply enough.

Dulness is also obtained frequently over the most prominent angularity of bone in the kyphoscoliotic thorax consistent with a healthy state of the subjacent lung.

Relatively dull sound as a normal quality occurs both as diminished vesicular resonance and diminished tympany. It results from percussing a solid organ having a small-sized or thin layer of air-containing structure between it and the chest wall, as where the lung overlays the heart and liver, or from percussion over air-containing tissue with a thin layer of airless tissue intervening, as where the lower border of the liver comes between the large bowel and the body wall. The former of these conditions gives rise to *normal vesicular dulness*, the latter to *normal tympanitic dulness*.

(B) **Intensity.**—This is simply *loudness*, and depends upon the amplitude of the vibrations, producing volume or mass of sound heard at varying distances.

A clear sound is always louder than a dull or dead sound; therefore, if in percussing over air-containing tissue of normal clearness and loudness we pass to parts that gradually or sharply diminish in loudness, we know that underneath lies more or less airless structure.

The intensity of the tympanitic sound is greater than that of lung resonance, volume for volume and other things being equal; for instance, a stomach distended with air or gas to the capacity of the left lung elicits a clearer and louder tone than the latter, where the structure consists of numerous little air vesicles formed by considerable spongy tissue instead of one large, free viscus. In disease of the lung, however, the intensity of the tympanitic note over a tubercular cavity is softer than over the surrounding vesicular substance, for the reason that smaller volume affords less amplitude, and hence less acoustic force, than larger volume.

The loudness of resonant sounds depends upon three conditions: (1) The force of the percussion stroke; (2) the thickness and elasticity of the chest wall; (3) the volume of the air-containing parts set in vibration.

(1) THE FORCE OF THE BLOW bears a direct relation; *ceteris paribus*, the stronger it is the greater the intensity, as in striking a drumhead; the loudness varies while the quality remains the same. Modifications of the strength of the percussion blow will be considered later in their application to delimiting organs, the lung borders especially.

(2) The greater the THICKNESS OF THE CHEST WALL the less the intensity; and, conversely, the nearer the air-containing organ to the finger percussing, the less the muffling of sound, provided the strokes are delivered with equal force. Elasticity of the thoracic framework enhances the intensity.

(3) THE AMOUNT OF AIR-CONTAINING TISSUE SET IN MOTION influences loudness in direct ratio; the greater the amount the louder the sound, and *vice versa*. That is, using the drum as illustration, with equal strength of blows and thickness of membrane, the sound of bass is louder than that of small or kettle-drum.

(C) **Pitch.**—In music, pitch is the elevation or depression, the acuteness or gravity of the scale notes, depending upon the rate or rapidity of the vibrations per unit (second) of time. The more rapid the vibrations the higher the pitch; at the same time they are shorter in wave length than those which produce the lower tones. Percussion sounds vary so in pitch, even in the normal, that only the talented or cultivated ear, or both, suffices to give the practised familiarity to detect the slight differences of pitch of diagnostic or prognostic significance. Changes in quality are more readily appreciated, but as these are often recognized by this very attribute of pitch, sharpness in the sensitiveness to fine degrees of the latter adds immensely to the early discovery of incipient pulmonary disease.

Pitch is the most important element in the differential diagnosis of airless from air-containing tissue, and in outlining by percussion the borders of adjacent or overlapping organs of clear and dull sounds, as the lungs from the heart or liver, spleen from stomach, etc. The transition from clearness to deadness is realized more by the gradual elevation of pitch than by any other acoustic attribute,

for the less the amount of air in the lung the duller—the noisier—the sound and the higher the pitch.

The relation of intensity to pitch is this: The louder or clearer the sound the lower the pitch; the higher or shriller the sound the less its loudness. Again the illustration of the large and small drums holds: the low-pitched bass drum emits a louder, farther-carrying sound than the higher-pitched kettle-drum.

Similarly, a *clear* sound has a *low pitch*, and is consequently *louder* than a *dull* one, which has a comparatively *high pitch*.

A word of *caution* is needed here to the student, namely, not to confuse the high-pitched, dull sound with the high-pitched tympanitic note often met with over small cavities of the lung, for example.

Other factors being equal, the pitch of the *tympanitic* tone is lower—and at the same time louder—than the *pulmonary resonance*. Practically, however, as far as size is concerned, only the occurrence of a dilated stomach affords a demonstration of this physical fact. This leads to the very important

CONDITIONS THAT INFLUENCE THE PRODUCTION AND VARIATION OF PITCH.—In general, the larger the cavity containing air the lower the pitch; therefore, owing to the *volume* of the lungs, the pitch of the normal percussion sound is distinctly low over them. As the vesicular tissue is elastic, however, the degree of tension is also a factor influencing the pitch both physiologically and pathologically in respiratory conditions. Ordinarily the lung vesicles are not in a state of high tension; therefore, if the tension is increased and the vesicles forcibly distended, the pitch is raised, as at the end of a forced inspiration; or again, at the end of a deep expiration, the vesicles being relaxed, the pitch is relatively lowered. In the former case we hear what Flint termed *vesiculotympanitic resonance*; in the latter, the sound is exaggerated a little in clearness and loudness, and becomes ***hyper-resonance***. Whenever, as in certain diseased conditions that will be referred to later, the degree of relaxation becomes so marked that the vesicles are in a state of elastic equilibrium—the inspiratory and expiratory pressure being about equal—tympanitic quality is elicited with still lower-pitched and louder sound, as when healthy lung, removed from the body, is percussed.

It has been affirmed by some clinicians that, contrary to what has just been said, the pitch is lowered with the increased tension of the vesicular tissue at the height of inspiration. This apparent contradiction is due to not taking into account the conditions of expansion and the volume of lung in certain individuals in relation to the tension. To begin with, as an independent factor, and other things being alike, the presumption is in favor of the explanation first given, since it is in harmony with a law of physics that the greater the tension of a membrane the higher the pitch. Also, it is true that the larger the volume the lower the pitch is. Hence, the whole matter resolves itself into a question of the relative predominance of one or other condition with either portion of the respiratory act. That is, if in one case the expansive capacity of the lungs is so marked that with a full inspiration the volume of air-containing tissue increases relatively more than the tension of its walls, then the pitch will be lower than with ordinary quiet breathing. On the other hand, in a chest with feebler power of respiratory expansion, the tension may be increased out of all proportion to the increase of volume, which would result in an inspiratory elevation of pitch. This exposition is given here not so much because of any practical importance, but because the student may more thoroughly and clearly grasp the principles involved, for wider application later, by thinking out such a fundamental problem in physical diagnosis as this is.

The *influence of the chest wall* on pitch is practically confined to its thickness or density, or to its tension. Increase of these elements produces higher pitch. Indeed, it is probable that the tension of the chest wall is more important in determining the degree of pitch than that of the lung substance.

The *pitch of the normal lung resonance*, then, as compared with the conditions ordinarily met with, is *low*; at the *end of a forced expiration* it is a *trifle lower*; with a still greater (abnormal) diminution of tension the resonance becomes *deeper and clearer—hyperresonance*; lastly, when the lung is markedly *relaxed* and *retracted* the note becomes actually *tympanitic* in quality, with *lowest pitch*. Hyperresonance occurs also in *emphysema*, where the threefold condition of increased volume and decreased tension (loss of elasticity) of the vesicular tissue and the enlargement of the ribs contribute and combine to produce the abnormally deep, almost tympanitic sound or characteristic "bandbox" note.

The *pitch of tympanitic sounds* varies according to the appended factors. Of the *open* tympanitic sound the pitch is determined by: (*a*) The *size of the communicating opening*; the larger it is the higher the pitch; (*b*) the *volume of the air-containing cavity*; the larger the size the lower the tone; (*c*) the *tension of the walls*, if resilient; the greater the tension the higher the pitch.

Over *closed* cavities the pitch of the tympanitic note depends upon:

(*a*) The *volume* of the cavity; (*b*) the *tension* of its yielding walls: the same as *b* and *c* preceding.

Of the two varieties of tympanicity the pitch of the open is usually higher than that of the closed; at the same time the open tone is more distinctly drumlike in quality.

The tympanitic sound, whether open or closed in quality, is never found in percussing over the normal chest, but as a physical sign of pathological cavities of the lung its occurrence is not at all infrequent. However, good examples of the characteristics of the open, high-pitched tympanicity are heard in percussing or filliping the larynx or the cheeks, causing a sort of "tubular" note. When the mouth is closed a lower pitch is detected, giving rise to the timbre known as *amphoric resonance.* Still graver tones are noticed in eliciting the closed tympanicity over the bowels, and gravest of all over the moderately air- or gas-distended stomach—pronounced hollowness.

When the cavities are *cylindrical* and communicate with the external air, the pitch depends upon the *length of the tube*; the longer it is the higher the tone. This may be demonstrated satisfactorily by percussing lightly over a long-necked flask, successively empty, half- and nearly filled with water.

As regards the influence of *volume* upon pitch, there are many gradations from the loud, low stomach sound to the high-pitched, open tympanitic note over a small lung cavity (tuberculous).

The pitch of tympanicity as affected by the *tension* of the cavity walls is of some importance. Extreme tension may destroy the tympanitic quality. It has already been remarked that when the tension of the lungs is greatly diminished, whether pathologically, as by the encroachment of a pleuritic exudation, or when the lungs in a healthy state are removed from the body, the resonance becomes tympanitic. Just so, on the other hand, may the tension of the walls be exaggerated so that tympanicity is lost and clear nontympanitic or lung sound be produced in the case of cavities or hollow organs. Thus, we hear the pulmonary vesicular quality of sound in percussing over stomach and intestines that are forcibly distended with air or gas. As soon as the internal pressure is relieved the tympanitic character is again restored. In the living subject, therefore, the lower tension and pitch of normal stomach and bowels and abnormally relaxed lung correspond; while the normally inflated lungs and abnormally distended gastro-intestinal canal correspond in possessing both higher tension and higher pitch, the first group yielding tympanitic sound, the other nontympanitic resonance. In the case of the former, the air or gas alone is set into vibration, the flaccid walls tending to increase the amplitude of the sound waves by reflecting them; in the latter, not merely the contained air—packed in and condensed, as it were—vibrates to the percussion stroke as best it may, but the tense mural tissues also respond with short, accelerated waves of higher pitch and change of quality because of the commingling of the vibrations of this solid substance. Instead of the consonance of the reflected and intensified waves of tympany, we have the more dissonant, resisting, interference waves of the pulmonary type of resonance. Enormously tight distention may give rise to such inability of the contained gas to vibrate that the possible clearness of sound due to its presence is quite neutralized by the vibrations of its containing walls only; hence the dull sound of solid tissue, with corresponding elevation of pitch.

The *pitch of dull sounds* varies according to the degree of density of the tissue percussed; the denser it is, the duller or flatter the quality, the higher the pitch. In other words, pathologic diminution of air-containing tissue is indicated whenever, in percussing over any region of the chest, for example, in either infraclavicular space, the pitch is found to be distinctly elevated as compared with the same region on the other side, or with adjacent normal vesicular substance; in fact, the detection of airless tissue abnormally located, by the evidence of dulness, is made essentially by the attribute of comparatively higher pitch.

In a general way it may be stated, that any pathologic condition which adds dulness to or impairs the clearness of a resonant or tympanitic sound, thereby raises its pitch and also reduces its loudness.

Normally, the proximity of the heart and liver—airless organs—to the borders of the lungs muffles or deadens slightly the clearness obtained in percussing over the latter, and this is recognized by the slight rise in pitch—relative dulness.

It should be remembered, however, that a high-pitched sound is not necessarily a dull one, as it may be tympanitic from a small vomica (cavity) in the lung, or resonant, as over the tip of the lung apex posteriorly, above the scapula.

Attention may be called here to the very slightly higher pitch elicited on percussing over the right lung as compared with the left, due to the difference of density which causes the normal vocal fremitus to be a trifle more marked here also (see Palpation).

(D) **Duration.**—This attribute—the length of time a sound can be heard—is the least important of the four, but in combination with the others may be a very helpful element in determining small variations of sound characteristics:

The sound vibrations developed by percussing over normal lung remain audible distinctly longer than over the liver or heart, and least so in striking the thigh. Thus duration varies directly with intensity and clearness; the louder and clearer the tone, also the lower the pitch, the longer the duration, the less loud, duller, higher-pitched sounds having relatively short duration.

The normal pulmonary resonance being full and clear, loud, and grave in pitch, has long duration. The sonorous tympanitic sound is likewise lengthily audible, depending upon the volume and pitch; if produced by percussing a large, dilated, gas-filled stomach, the duration will be greater than the lung resonance. Obviously, too, the duration of a relatively dull note is longer than an absolutely dull or flat sound; the conditions which influence the production of persistent sounds are the same as those which affect their clearness, loudness, and graveness, and *vice versa.*

(E) **Sensation of Resistance.**—This has been alluded to under palpatory percussion. While percussing over air-containing organs, as the stomach and lungs, and then over an airless organ, as the liver, a feeling of resistance by the striking as well as the pleximeter finger is noticed over the latter. Thus, clear sounds are associated with slight resistance, and dull sounds with a decided resistance.

This sign bears a direct ratio to pitch; that is, as the pitch rises the resistance is felt to increase. It depends upon the *degree of capacity of the underlying parts to vibrate*; upon their consistence and thickness. Normally, the sense of yielding or elasticity is much greater over the interspaces than over the ribs or scapulæ. Pathologically, lung conditions producing dead or dull sounds, as large consolidations or large pleuritic exudations and the like, have decreased vibrating capacity, and consequently offer great resistance to the percussing fingers; this is intensified if there is accompanying increased tension of the chest wall, while conditions

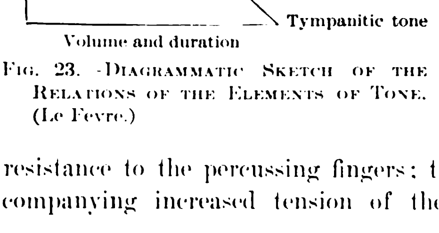


Fig. 23.—Diagrammatic Sketch of the Relations of the Elements of Tone. (Le Fevre.)

which augment the amount of air, provided mural tension is not increased at the same time, give greater resiliency and less resistance.

To parallel the ***associated attributes*** of sound as they occur for the recognition of the two extremes of gross physical conditions and changes of the bodily organs, the following is easily apprehended:

	Air-containing Structures.	**Airless or Solid Structures.**
Sounds:	*Clear* in quality,	*Dull*, dead, or flat.
	Loud in intensity,	*Not loud.*
	Low in pitch,	*High-pitched.*
	Long in duration,	*Short.*
Sensation of resistance:	*Slight, absent,*	*Marked.*

PERCUSSION SOUNDS OF THE NORMAL CHEST

Regional Variations

Prerequisite to any practical knowledge and judgment concerning pathologic alterations of the percussion sounds, and of the regional and anatomical limits over which they are audible, is a retentive and ready knowledge of the normal boundaries, and of the anatomic and physiologic differences of sound over the principal areas of the healthy lungs.

Technic of Percussion of the Lungs.—Using the most serviceable —the finger—method, we begin in front to percuss the apices in the SUPRACLAVICULAR SPACES, carefully comparing one side with the other repeatedly, at the end of or during a full inspiration and a forced expiration. The strokes should be delivered with moderate force. It will be noticed that the apex of the *right* lung is not so resonant as the left; that is, the pitch over the right apex is a trifle higher than over the left. This corresponds to the slightly exaggerated but normally greater vocal fremitus over the right side.

A slightly less resonant sound over the left apex, as from a small tubercular consolidation, might not differ in quality from that elicited over the normal right apex; hence, the discovery of slightly decreased resonance (and elevated pitch) over the left top is presumptive evidence of the presence of a pathologic enfeeblement there, even when these signs are no more pronounced than over the right corresponding region. If, instead of the slight normal, there occurs a decided lessening of the resonance over the right apex, then its pathologic indication is clear, although its occurrence on the left side would be all the stronger evidence of disease there.

The predominating frequency with which the apex of either lung becomes the seat of an incipient tuberculous process necessitates especial care in the percussion of presumably early cases. Familiarity with the physical signs of the apical region and their variations in health is the indispensable prerequisite in detecting incipient disease. As pointed out by Minor, Auld, Landolfi, and many others, the resonant area above the clavicles should be carefully outlined comparatively with and without respect to deep inspiration.

Early indications of abnormalities are (1) failure of the upper line of resonance on one or the other side to ascend during a deep inspiration; (2) a lowering of the upper line of resonance, in whole or in part, as compared with that of the opposite side; and (3) indistinct definition of the upper or outer line of resonance.

Landolfi (*Semaine Medicale*, XXVI, No. 11), of Naples, has indicated six points (demonstrated by subsequent autopsies) where percussion is liable to reveal impaired resonance suspicious of an incipient tuberculous process. Point 1 is 1 cm. below the clavicle, at the junction of the inner third and the outer two-thirds. Point 2 is the same distance above the clavicle, on the same vertical line. Point 3 is at the intersection of the acromiomastoid line with a line uniting Point 1 with Point 4. The latter point is in the center of a line drawn from the acromion to the spinous process of the second dorsal vertebra, and is the only point on the back. Point 5 is 1 cm. below the center of the clavicle, and Point 6 is just inside the acromion and above the acromiocervical line. He points out that the less the difference between the results of light and heavy percussion at Point 6 the greater the probability of a lesion of the apex.

Near the inner end of the clavicle the vesicular resonance becomes a little tympanitic because of the proximity of the trachea. The pitch is slightly lower at the acromial than at the sternal end of the clavicle.

Immediate percussion of the CLAVICLES produces a clear sound of the mixed pulmonary and bone elements. As the character of the resonance will vary with the size and shape of the bone, the true condition of the lung underneath is not always indicated. The irregular and calloused form of a previously fractured clavicle would be particularly misleading in either direct or comparative observations.

On the STERNUM the note is clear and deeply resonant, with a slightly tympanitic quality over the manubrium, on account of the nearness of the trachea and roots of the bronchi. The resonance down to the third rib is fully vesicular; below that it is a trifle dull because of the adjacent influence of the heart (right ventricle) and liver (left lobe). As pointed out before, though lung tissue does not lie directly behind the sternum, the clear sound is produced by the vibrations setting into acoustic activity the neighboring lung. Along the left edge of the sternum, between the fourth and sixth ribs, the heart, uncovered by lung, renders a dull percussion sound.

Next the INFRACLAVICULAR SPACES are percussed, comparing the first and second interspaces on each side, using moderately strong strokes. Here we obtain the typical lung or vesicular resonance—the standard region for each individual—again noting, however, the very slightly duller, higher-pitched, shorter sound on the right side. Thus individual chests have their individual degrees of resonance, the normal, regional deviations from which must be estimated with the best judgment possible for each person, adopting the note elicited in the second interspace, below the middle of the clavicle, as a sort of individual resonant keynote.

The vesicular quality is less distinct in percussing near the sternum on account of the muffled tympanitic modification due to the adjacent sternum and bronchotracheal tubes. From the midclavicular (mammillary) line outward the resonance is a trifle less clear and deep.

Three explanations are usually given for the slightly duller pulmonary resonance on the right side, any one or all of which may cause the difference: (1) The greater thickness of the chest wall consequent upon the greater development of the pectoral muscles on the right side. This difference may be less apparent in the chests of left-handed persons, but not necessarily always so. (2) An explicit reason is found in the fact that there is a different anatomical arrangement of the bronchial tubes, that on the right side being larger, and situated more superficially and higher up; this, with a bunch of medium- and small-sized bronchial branches occupying space that on the left side is filled with air-vesicles, gives an amount of bronchovesicular tissue sufficient to elevate the pitch slightly but perceptibly, the tubes adding an element of tympany, the extra muscular and connective tissue of their firm walls an element of dulness. (3) Finally, the fact that the right lung rests upon a solid organ—the liver—may influence the resonance enough to impair its vesicular clearness, loudness, and depth of pitch.

Mammary Regions.—Beginning at the third interspace and percussing down the nipple line on the *right* side, we find the pulmonary resonance becoming gradually relatively dull, just noticeable in the fourth interspace owing to the increased thickness of the chest wall from adipose tissue and, especially in women, the mammæ, besides the approach to the dome of the liver and the thinning of the interposed layer of lung, until the fifth interspace is reached, where the thin border of lung is only sufficient to prevent the relative dulness from being absolutely dull, as it is in the sixth interspace with light percussion, and more decidedly in the seventh and eighth interspaces with heavier percussion. Strong strokes are necessary where the chest wall seems to be thickest in order to bring out the acoustic evidence of the subjacent air-cells.

On the *left* side, slight relative dulness begins in the third interspace, in the parasternal line, with moderately strong plexor blows. Here there is but a thin layer of lung resting over the upper and outer borders of the heart, and gliding to and fro with inspiration and expiration. In the fourth and fifth interspaces pulmonary resonance is entirely replaced by absolute cardiac dulness, readily detected with light percussion blows. Outside the left mammillary line, as far as the anterior axillary line, and in the fifth and sixth interspaces, there is a degree of resonance a little less pronounced than over the space above, at times modified slightly at the lower and outer parts by the mingling of stomach tympany. Of course, the cardiac dulness and gastric tympany are both less distinct when the inflated lung is percussed at the end of a full inspiration.

Over the *right inframammary region* we meet with absolute liver dulness from the sixth rib down to the costal border; in approaching the latter, however, the percussion strokes must not be given with much force or the tympanitic sound of the large intestine below will be elicited and overshadow the dulness before the edge of the liver has actually been reached. The sensation of resistance here is quite marked.

On the *left* side the sound obtained is mainly tympanitic on account of the stomach; if not distended with air or gas, the stomach sound may be a muffled tympany, or if after a meal percussion is made here, the note will be dull, this quality being contributed to by the adjacent left lobe of the liver above and to the right, and the spleen to the left. This tympanitic area, bounded above by the pulmonary resonance at the sixth rib, below by the slightly higher-pitched tympanicity of the splenic flexure of the colon, and to the right and left by the hepatic and splenic dulnesses just mentioned, is called often *"Traube's semilunar space."*

The *axillary regions* may be percussed next. On both sides the vesicular resonance is clear and full down to the seventh rib in the midaxillary line, but slightly impaired in the seventh interspace. In both infra-axillary regions below the eighth rib the sounds are dull; a little duller on the right side because of the liver; while on the left side there is part tympany and part dulness on account of the proximity respectively of the stomach and spleen.

Finally, we percuss the *posterior regions* of the thorax with the subject leaning forward a little, the arms folded, and all muscular contractions avoided as much as possible.

SUPRASCAPULAR SPACES.—Forcible percussion here yields pulmonary resonance of less intensity and higher pitch than in front, owing to the thickness of the intervening tissues and the very small volume of underlying air-cells. The tip of each apex is just below the flat surface of the supraspinous region, and an important point in searching for the evidences of incipient tuberculosis. The border of the trapezius muscle forms the dividing line of two conjoined triangles within which the anterior and posterior aspects of the lung apices may be examined. The lower side of the anterior triangle is formed by the clavicle, of the posterior by the spine of the scapula, both triangles converging at the point of the shoulder.

In the *scapular regions* strong percussion is required also to bring out even moderately the resonant tone, the thickness of bone and muscle interfering most decidedly with clearness, loudness, and graveness of pitch, as compared with the resonance obtained in the infraclavicular region, and resisting most markedly the plexor and pleximeter fingers. The spine of the scapula responds with a high-pitched osteal sound.

The resonance in the *interscapular region*, while better than over the supra- and infraspinous areas, is still weak, relatively, owing to the firm and thick musculature near the vertebral column. In the upper portion the trachea may superadd a tympanitic quality to the resonance.

The *infrascapular regions* emit the best resonance posteriorly during a moderate strength of percussion. From the angles of the scapulæ to the ninth interspaces the note is clear and of distinct resonant quality, although a little less so on the *right* side because of the larger muscular development in most people, and the subjacent solid liver. With heavy percussion relative dulness may be elicited as high as the eighth rib. Below the tenth rib absolute liver dulness is noted. On the *left* side, forcible blows may impart a tympanitic character from the stomach and colon beneath, or a dull modification near the posterior axillary line because of the spleen.

To *summarize*: The pulmonary resonance is heard most typically in the left infraclavicular region; anteriorly, it is better heard over the upper half of the chest; posteriorly, over the lower half; it is more distinct and full anteriorly than laterally (axillæ), and more so laterally than posteriorly.

Regional Differences of Pitch.—These vary with the conditions of volume and tension of air-containing tissue, and of chest thickness and density already instanced. They practically mark the areas of different degrees of clear quality before given, for it is by pitch more than any other attribute that we note the regional changes and points of transition from one sound characteristic to another.

Thus, a rising gradation of pitch may be perceived in passing over the following anterolateral regions: grave pitch in Traube's semilunar space (overdistended stomach); axillary region (stomach and lung sound); the second interspace; the supraclavicular space; the third interspace (left); the fourth interspace (left); the seventh and eighth interspaces in the right midclavicular line (liver).

It is important to repeat in other and more general words that since comparative percussion must habitually be practised in the detection of disease, the normal variations of sound over symmetrical parts of the chest on the two sides should constantly be borne in mind. The most marked *dissimilarity* in healthy individuals is in the right and left mammary regions—the *neighborhood of the heart.* Further than this, it is only needful to remember especially the slightly clearer note over the left as compared with the right apex of the lungs, and generally, the fact that elsewhere on the right side the corresponding situations are a trifle less clear and intense than on the left.

Variations Due to Age and Sex.—The percussion sound differs normally according to *age.* In children, with their thin, elastic chest walls, the note is more resonant, graver in pitch, louder, and of longer duration than in aged persons; the quality is peculiarly soft and clear. The stiffness of the thorax and hardness of the ribs in old age, together with senile relaxation of the lungs, causes the resonance to be less pronounced and higher in pitch. This difference is apt to be all the more marked the thicker the fleshy coverings of the chest wall and the greater the diminution of lung volume. On the other hand, the change in pitch will be counteracted somewhat by emaciation of the chest and senile atrophy of the pulmonary parenchyma.

As to *sex*, we usually find the resonance to be more distinct in women than in men, especially in the upper regions, owing to the superior costal breathing and less firm and massive bony thorax.

Topographic Percussion

Determining the Boundaries of the Lungs and Adjacent Organs. TECHNIC.—As for diagnostic purposes it is important to ascertain the *size* of the lungs, the percutory determination of their borders on the surface of the thorax is a matter calling for careful practise. The position of the lung boundaries, as affected by physiologic and pathologic mobility, and the encroachments of other organs or of new growths, are also involved.

CONDITIONS.—In order that the borders of the lungs (or of any of the viscera) may be outlined topographically by percussion, it is necessary, in the first place, that they be sufficiently *parietal*; secondly, that they yield *sounds differing* from the surrounding and adjacent tissues. Hence, the boundaries between two organs giving forth absolutely dull sounds cannot be recognized, as between the inferior border of the heart and the left lobe of the liver, or between the left lower border of the heart and a left-sided pleural effusion. But the adjacent borders of an air-containing and an airless organ are the more readily determined; thus, the lung-heart and the lung-liver and the lung-spleen boundaries are found. Again, we can tell an organ giving a clear, tympanitic sound from one that causes a deadened sound, as stomach from spleen or liver; and yet again, clear sounds differing from each other in quality on the one hand, and pitch on the other; thus, lung from stomach in the case of the former, and stomach from bowel, or large from small bowel, in the case of the latter.

METHOD.—In determining the boundaries by percussion we usually percuss from clear to dull, no matter what the organ under examination, as the modifications of slight deadening are more quickly detected as affecting a resonance than those of clearness in passing from a dull area. Approach to an organ is made along lines perpendicular to its supposed borders, with the pleximeter finger consequently parallel to such borders. We percuss on these lines until the sound changes, striking at intervals of an inch or an interspace approximately at first, then repeating carefully and closely so that we are certain that at a given point lung ends and another organ begins; this point may be marked with a blue pencil, ink, or iodine dot or dash. Similar markings made along the boundaries so determined may be joined by a continuous line, which will then represent as nearly as possible the surface outline of the lungs, for example.

Accuracy in topographic percussion is better assured if it be made the rule to *percuss very lightly along the borders of the organ whose location and size we are essaying to define.*

Practising the contrary of this, by forcible strokes near the edge of organs, as of lung near the cardiac or hepatic borders, must obviously cause dulness to be elicited before one actually has passed beyond the lung boundary; or again, by strong percussion over the lower border of the liver, the adjacent underlying intestine is set in vibration, and a tympanitic tone is obtained before the edge of the liver has been reached.

Furthermore, as the volume of air-containing tissue of the lung borders is very slight, the sound must have little intensity and distinctive clearness; slight differences of sound on the border-line are better perceived if the sounds themselves are slight (Vierordt).

Normal Percussion Limits of the Lung.—The normal anatomic boundaries have already been described, but it is not possible to define them throughout by percussion, as the differences of sound are often slight, particularly at the left inferior border, near the stomach, the tympanitic sound of which frequently mingles with pulmonary resonance above the anatomic lung boundaries to confuse the real line of transition.

Also, the absence of the condition of adjacent differences of sound prevents us from delimiting the *anterior* borders of the lungs behind the sternum because of their parallel proximity, and the exaggerated uniformity of resonance produced by the characteristic osteal tone of the sternum itself.

The determinable percussion outlines of the lungs are, then, the *superior*, *cardiopulmonary*, and *inferior*.

(1) *The Apices.*—In healthy persons, during ordinary, quiet respiration, the vesicular resonance extends about 2 to 4 cm. (¾ to 1¾ in.) above the upper border of the clavicle. With a deep inspiration the apical resonance in the supraclavicular spaces is clearer and fuller, and more extensive in all directions. The determination of the upper borders of the lungs is important because of the evidence of tuberculous deposit or pleuritic adhesion which the discovery of unilateral depression or shrinking of the apex indicates.

In children suffering with membranous croup or extensive bronchopneumonitis, the accompanying collapse of the apical vesicles which may ensue prevents one's getting resonance above the clavicles.

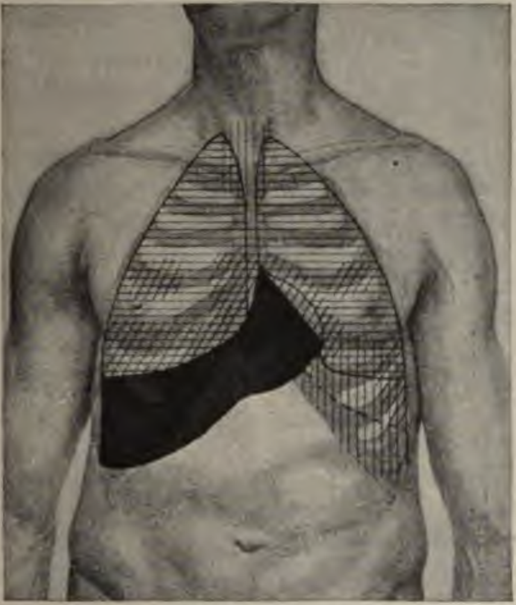


FIG. 24.—SHOWING THE RELATIVE RESONANCE OF VARIOUS PORTIONS OF THE ANTERIOR SURFACE OF THE THORAX. Horizontal lines = pulmonary resonance. Vertical lines = tympanitic resonance of trachea and stomach. Oblique lines = impaired resonance or moderate dulness due to mammary glands, liver, heart, and spleen. Solid shading = absolute dulness due to liver and heart. (Butler.)

Temporarily during attacks of bronchial asthma, and constantly in cases of emphysema, the superior border of the lungs is found on percussion to be higher than normal *with expiration*, owing to the difficult and delayed and incomplete expulsion of the tidal air as the thoracic expiratory movement takes place. At the same time the sound has a vesiculotympanitic quality.

(2) *The Lung-Heart Boundary.*—This is determinable only by light percussion, because of the small volume of lung at its border. It is found to follow along the fourth rib, from the sternum to a little external of the parasternal line, whence it passes downward and outward to the sixth rib in the midclavicular line, forming the upper and outer boundaries of the superficial cardiac dulness due to the exposed part of the right ventricle, and producing a corresponding

zone of impaired lung resonance (relative dulness) about the width of an interspace beyond the edge of the lung.

When the anterior border of the lung is physiologically distended with a full inspiration, or pathologically from emphysema, the encroachment it makes upon the cardiac dulness diminishes its area, and in the case of a marked emphysema may obliterate it entirely.

On the other hand, the percutory determination of retracted lung border may indicate tuberculous or fibroid disease, pleural effusion with collapse of lung, or displacement outward of this border by the pressure of a pericardial effusion or of an enlarged heart; the area of absolute cardiac dulness is correspondingly increased.

(3) *The Lower Borders.*—In front, the percussion limit of the lung does not extend quite as far down on the right as on the left side, the right coming low as the inferior border of the fifth rib, the left to the superior border of the sixth.

On the *right* side, in percussing down to the lung-liver boundary, a relatively dull sound is detected in the nipple line at the fifth rib approach, which passes into absolute or superficial liver dulness in the sixth interspace. The narrow zone of deep or relative liver dulness extends around the chest to the spinal column, between the seventh and eighth ribs in the axillary region and between the ninth and tenth ribs in the scapular line.

There may be an extension more or less beyond an interspace farther, due to emphysematous enlargement of the lung. Also, if the lower borders are higher than normal, we must think of the possibility of fibroid retraction, causing apparent or spurious enlargement of the liver, or a pushed-up diaphragm due to meteorism, ascites, large abdominal tumors, especially of the liver itself, and paralysis of the phrenic nerve.

Posteriorly, the lower border of the lung may be pushed up by a pleural effusion, the dulness produced by which extending upward continuous with that of the liver joins a modified—more or less tympanitic—resonance where the partially collapsed lung is reached.

On the *left* side, percussion downward to the inferior border at the sixth rib in the mammillary line is resonant, but along the border to the midaxillary line it is slightly admixed with the tympanitic element—the *lung-stomach boundary.* At the eighth rib and midaxillary line we note slightly duller note of the *lung-spleen boundary.* Finally, near the spine, we define less distinctly the *lung-kidney boundary,* at the level of the tenth interspace or eleventh rib.

Differences Due to Age.—In *young children* the lower borders of the lungs are from one-half to one inch—about an interspace—higher than for the normal person of middle age; in the *old,* the borders extend a rib or interspace lower, the lungs increasing in size with advancing years. For the same reason, the lung-heart boundary leaves less heart area uncovered by lung.

In estimating the percussion limits of the lower borders of the lungs, allowance must also be made for their physiologic *displacements* as produced by the *respiratory* or *active mobility* and the *postural* or *passive mobility.*

(1) *Respiratory Mobility.*—In quiet breathing the lower borders extend only about ⅜ in. (1 cm.) into the inferior complementary pleural sinus (q. v.), from 1¼ to 1½ in. (3 to 4 cm.) in deep inspiration in the midaxillary line, and about ¾ to 1¼ in. (2 to 3 cm.) in the midclavicular and scapular lines. According to Weil, in deepest expiration the lung borders move a little less than the same extent above the average location. The mobility of the lung edges where they overlap the heart is also a trifle less than inferiorly.

(2) *Postural Mobility.*—The respiratory displacement depends upon the position of the body. In the *dorsal* position the anterior lower edge of the lung moves ¾ in. (2 cm.) lower than in the vertical position. On turning from the back to the (either) *side,* while recumbent, the lower border descends as much as 4 in. (10 cm.) in the midaxillary line, on the uppermost side, although a mobility of but 2 in. on the part of the uppermost lung is more common.

The pathologic alterations of the topographic pulmonary outlines (already referred to) will be considered also in connection with their relations to the abnormalities of active and passive mobility, after the description of the diagnostic significance of pathologic percussion sounds given in the next chapter.

CHAPTER V

PERCUSSION (*Concluded*)

THORACIC PERCUSSION SOUNDS (Concluded)

ABNORMAL PERCUSSION SOUNDS DUE TO DISEASES OF THE LUNGS AND PLEURÆ

Their Physical Explanation and Diagnostic Significance

WHILE a certain and constantly improving skill in technic, and **perception and judgment in regard to** the normal and regional variations **of the lung sound are prerequisites** in this method of examination, **it is the main object to seek for** the abnormal—either the detection **of a normally occurring sound** in an abnormal location or of **alterations of normal sounds in any** location—and then to know the physical and pathologic conditions which give rise to them, with their diagnostic interpretation.

The immediate aim and result of percussion is to determine the density of the subjacent parts. Inferentially, that means, *first,* the determination of any increase or diminution of the normal quantity of air present in the lungs; and, *secondly,* the delimitation of the site and area within which the change has occurred, including the differentiation of pathologic lung boundaries from adjacent air-containing and airless organs.

Thoracic percussion is also an important aid in ascertaining abnormal physical conditions of the pleural membranes and sacs.

Abnormal sound over the lungs may indicate not only the presence or absence and the volume of air, but changes in the quantity, density, and tension of the pulmonary tissue, as will be shown later.

There may be areas over both lungs which yield percussion sounds different from the normal, hence the presence of ***bilateral*** disease, or they may be found only on one side, in ***unilateral*** disease; finally, there may be but one or two small areas of ***local*** involvement.

A. *Dulness: Impaired or Deadened Resonance*

Pathologic dull sounds may be comparatively slight, in relation to the corresponding region on the other side, whence the terms *impaired, diminished*, or *deficient resonance*, or *slight dulness*; or there may be *moderate, decided*, or *marked dulness*; lastly, when the part gives forth a sound like that over the thigh or over a solid organ, it is termed absolutely *dull, dead*, or *flat*.

When dulness is caused by disease within the lung, the condition is *intrapulmonary*; when caused by pleural affections, *extrapulmonary*.

Intrapulmonary Conditions.—Resonance is deadened here by the deposit or infiltration of airless tissue in the lung, causing consolidation and compression.

We recall, also, besides the change in the quality of the percussion note, the diminished intensity, raised pitch, and lessened duration.

(1) SLIGHT DULNESS.—This occurs where the air vesicles are blocked or consolidated in *small, lobular areas*. The location and number of such areas of consolidation may be of aid in diagnosis. Thus, if located in front, at or near the apices, and single in occurrence, it usually indicates tuberculous deposit there; if lower down, posteriorly, and multiple in number, a bronchopneumonitis is usually indicated. Again, early tuberculosis is generally one-sided, while the latter is bilateral.

In order that the dulness may be detected over small consolidations, light percussion strokes are necessary, so as to avoid setting into vibration the closely surrounding open air vesicles. Where several small areas of consolidation exist closely adjacent to each other, the intervening vesicular tissue, being relaxed, elicits a high-pitched tympanitic sound, more or less muffled according to the nearness and largeness of the consolidated areas.

In *early tuberculosis of one apex*, very frequently the first and only physical evidence of apical consolidation is manifested by diminished resonance a little behind and below the border of the trapezius muscle, in the supraspinous region on the affected side.

Whether the unilateral consolidation be in the supraclavicular or supraspinous region, the resulting slight or relative dulness is more easily detected if located on the left than the right side, because of the slight normal impairment of resonance on the right side; so that a degree of dulness over the left apex equal to that over the right justifies the suspicion of disease on the left, while the degree of dulness on the right side sufficient to indicate consolidation there must be greater than the normal difference between the two sides; in other words, a slightly more advanced stage of deposit. Perforce, dulness that is distinctly, though slightly, greater on the left than on the right side is all the more positively significant of disease on the left side.

Whenever *both apices are diseased*, and comparison of one side with the other is thus inadmissible, the fact is demonstrated by comparing the sound with the resonance over the adjacent parts lower down on the same side.

In persons with sedentary habits and occupations who are poor breathers, and in those who may be thus, or by heredity, predisposed to pulmonary tuberculosis, the finding of slight impairment of resonance over both apices, disappearing after a few forced respirations, is evidence that such individuals do not ordinarily use their apices, which become more or less collapsed for want of air.

Small patches of slight dulness on percussion, in the interscapular region near the spinal column, may indicate the presence of *tuberculous enlarged bronchial glands*, or of accumulations of *exudation within the bronchial tubes.*

Hemorrhagic infarct of the lung may be inferred where a small area of slight dulness is discovered, especially over the middle and lower lobes of the right lung, after the sudden onset of respiratory distress where mitral valvular heart disease exists. The greater the exclusion of air, and its area being not less than 1½ in. (4 or 5 cm.) in diameter, the better the dulness.

Besides the occurrence of bronchopneumonitis, small areas of impaired resonance over the lower lobes posteriorly may be caused by *atelectasis,* as from the obstructive bronchial closures of childhood, and by *thickened and dilated bronchi,* and *solid growths* on or near the surface of the lung.

(2) MODERATE DULNESS.—This is present when there is moderate infiltration of the lung, as a small portion of a lobe. It is found, for example, in *tuberculous disease* of the apices when the consolidation has progressed beyond the first stages of just-recognizable deposit. It is also obtained over scattered areas of the *bronchopneumonitis,* acute and subacute or catarrhal, characteristic of old people, especially as a complication of influenza, these patches being large enough to make the circumscribed areas of dulness unmistakable enough so that the surrounding zones of relaxed, tympanitic lung do not markedly modify their tonelessness. These, with the patches of atelectasis which

often accompany them, are usually found over the lower lobes posteriorly.

Moderate dulness is present also posteriorly at the base in the congestion of *hypostasis of weak heart*, particularly when some exudation into the alveoli has taken place (*pulmonary edema*), as in those who, from prolonged and exhausting sickness, have had to remain most of the time in a dorsal position.

Abscess, gangrene, large hemorrhagic infarcts, and *tumors of the lung*, and *thick-walled bronchiectatic cavities* partially filled with exudate, also give rise to moderate percussion dulness.

Here should be considered the important relation between the strength of the percussion stroke and the depth of location of partial lobar consolidations of the lung.

The positiveness with which a partial consolidation of the lung is revealed by the relative or moderate dulness elicited depends upon whether it is near enough to the chest wall to be discovered with light or medium percussion force, or whether it is so far beneath the surface that it can be discovered only with the most forcible strokes permissible by the tolerance of the patient.

In other words, to put it in an inductive and practical way, if with a comparatively light percussion stroke a small or medium-sized area of relative dulness is found which, with stronger strokes, becomes louder and resonant, it betokens the presence of a superficial, subpleural, airless mass so thin that heavy strokes penetrate through it to set into vibration the air-containing vesicles below it. If, however, over a given area, light percussion blows yield clear, resonant sounds of normal quality for that region, while a decided strengthening of the blow over the same space yields a marked relative dulness, it means, obviously, that there is a deeply seated consolidation, with sufficient intervening normal or relaxed lung to prevent any but powerful percussion blows from reaching it so as to give the muffled, less loud, and higher-pitched note.

A parietal patch of consolidation may be recognized by dulness on light percussion if not smaller than about 1½ in. in diameter; larger consolidations may be discovered by forcible percussion if not more deeply seated than about 2½ to 3 in. (6 to 7 cm.).

(3) Absolute Dulness.—This is obtained when a large portion of the lung, as a whole lobe, or even the whole lung, has been rendered practically airless, solid. It is typically and commonly present in *acute lobar pneumonitis*, where the alveoli of one or more lobes are filled with a firm, fibrinous, inflammatory exudate in the stage of

hepatization (liver-like condition). And yet the sound is rarely as completely deadened as the thigh sound, but may frequently have a slight admixture of the tympanitic element because of adjacent portions of merely congested, relaxed lung. The sensation of resistance is decidedly increased.

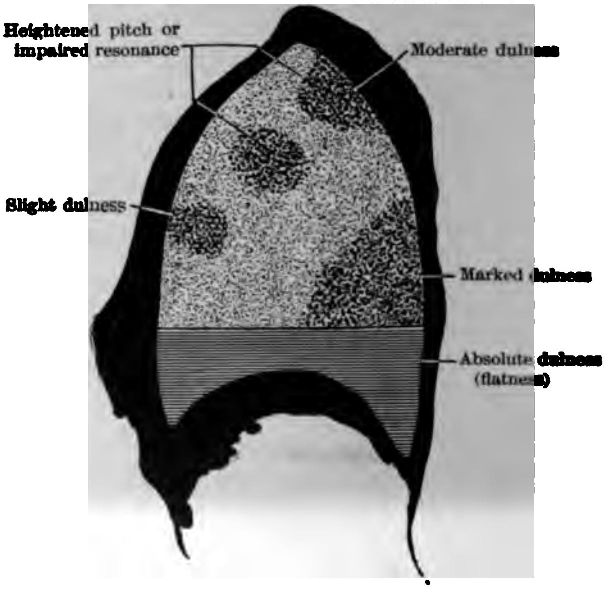

Heightened pitch or impaired resonance
Moderate dulness
Slight dulness
Marked dulness
Absolute dulness (flatness)

Fig. 25.—Showing the Variations of Lessened Resonance Due to the Presence of Consolidations in the Lung or Fluid in the Pleural Cavity. (Butler.)

If, in addition to the vesicular tissue, **the bronchial tubes within** the consolidated area are at the same time **clogged up with the exudation** (*massive pneumonia*), or if, as often **happens, there is marked** pleuritic exudation, either fibrinous or **serofibrinous, the dulness becomes** extreme or flat as a result of **such complications, with high** pitch, short duration of sound, and **intense resistance. As the lower** lobes are by far the most frequently **attacked in pneumonitis,** the dulness is generally limited to the **infrascapular and axillary regions,** and more often on the right than the left side.

Absolute dulness is also present over extensive **areas of** ***tubercular*** *consolidation*, with *thickening of the pleura*, **over the upper parts** of the chest.

Rarely, absolute dulness may be elicited **because of large** ***pulmonary*** *abscesses* and *mediastinal tumors* of **considerable size pushing** the lung aside and touching the chest wall.

The extent of the area of dulness in acute lobar (croupous) pneumonitis may coincide with the normal limits of the lobe affected, topographically; but as a pulmonary lobe, when completely consolidated, is more often considerably increased in bulk, the area of dulness is correspondingly enlarged. Thus, posteriorly the dulness may extend upward almost to the apex without the upper lobe being involved, and on the left side, with pneumonitis of the lower lobe, the encroachment of dulness downward upon the semilunar stomach space is noted in the diminished tympanitic area of the latter.

Finally, a peculiar but characteristic "wooden" quality of percussion dulness, occurring in the chests of certain old, emaciated, but "wiry" individuals who have had one or more attacks of pneumonitis or pleuritis, may be found, usually affecting nearly all of one side. I have seen such cases more often in miners and veterans of the Civil War. The sensation of resistance is much increased. The condition indicated is that of marked fibroid thickening in the lung; hence the terms *fibroid phthisis* and chronic interstitial pneumonia. Some thickening of the overlying pleura is generally present also.

Extrapulmonary Conditions.—The most important of these is *pleuritic exudation. Marked dulness* or flatness accompanies the presence of liquid effusion in the pleural sac, and also of *thickening of the pleura* due to plastic fibrinous deposit of sufficient amount. Even slight thickening of the pleura may be detected by moderate dulness, and increased resistance or hardness of "feel," on account of the contiguous nearness of the exudate to the intercostal tissues. Otherwise, the intensity of the deadness of sound depends upon the degree of thickening which may accompany the presence of fluid lower down or remain after the removal of the latter by absorption or aspiration with the hollow needle. Considerable dulness over the apices in early tuberculosis is frequently due to pleural thickening.

The amount of liquid exudate necessary to produce appreciable dulness at the base of the chest is about 400 c.c. This gravitating into the lowest and most superficial part of the complementary pleural sinus in the posterolateral region, and having a thickness of about three-quarters of an inch, may be recognized by light percussion, the dulness extending not more than two or three fingers'-breadth above the posterior lower limits of the lung. And when the layer measures more than two or three inches in depth, ordinary percussion elicits absolute dulness, any further augmentation of fluid causing extension of dulness from the base to the upper level.

As the quantity of liquid increases the area of dulness spreads laterally and upward, then to the front, and still higher, so that when the pleural sac is half or more than half filled we find the dulness extending from the vertebral column around to the sternum, and from the lower borders of the ribs to the upper intercostal spaces, though most decidedly flat at the base. Pleural effusion of the right side is recognized by an extension upward, apparently, of the liver dulness. On the left side, a diminution of the normal area of tympany in Traube's half-moon-shaped space, more pronounced than in cases of lobar pneumonitis; and the discovery of a band of dulness reaching upward in the anterior axillary region from the eighth rib to the sixth or higher, is characteristic of the presence of fluid here.

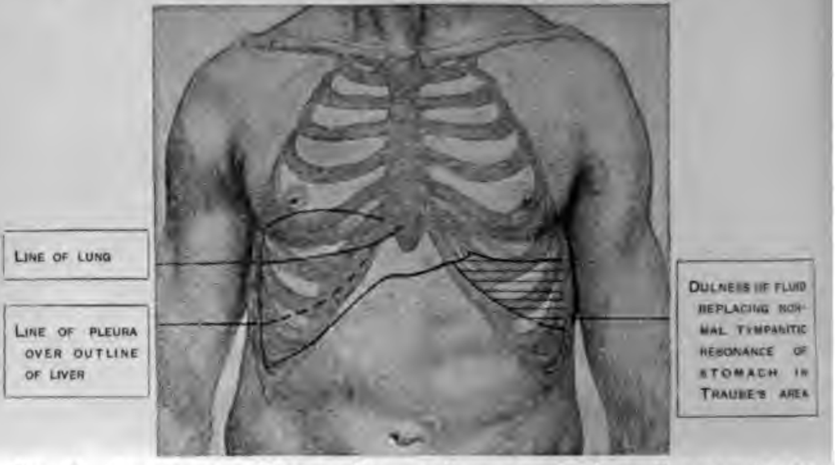

LINE OF LUNG
LINE OF PLEURA OVER OUTLINE OF LIVER
DULNESS OF FLUID REPLACING NORMAL TYMPANITIC RESONANCE OF STOMACH IN TRAUBE'S AREA

FIG. 26.—SHOWING THE DULNESS DUE TO FLUID IN THE LEFT COMPLEMENTARY (REFLECTED) PLEURA. (Butler.)

The upper level of a liquid exudation usually extends higher posteriorly than anterolaterally in patients confined to bed, the fluid being free to move; while in those who are able to be about, as in some subacute and chronic cases, the level is maintained nearly horizontal round the chest.

The dulness obtained in percussing over a pleural effusion is the result both of the intervention of a nonresonant substance and of the simultaneous proportionate expulsion of air from the adjacent compressed lung. What resonance is obtained even near the upper level of the exudate is very shallow because of the small volume of air present.

Grocco's Paravertebral Triangle in Pleural Effusions.—This sign is of importance in the diagnosis of one-sided pleural effusions. It was stated by Grocco, in 1902, that in case of a pleural effusion on one side, the other side presented a triangular area of dulness over the back of the thorax. The inner line of this triangle runs along the spine, the lower line coincides with the lower margin of the resonance for 3 to 10 cm., and the outer line of the triangle follows an oblique line, uniting at an acute angle with the first line on a level with the top of the effusion. This paravertebral dulness was always more marked when the effusion was on the right side. The proof of the genuineness of the triangular dulness is its disappearance when the patient lies on the side of the effusion, and its reappearance immediately he sits up or lies upon the sound side. When the pleura is much distended the mobility of the fluid is reduced, but the paravertebral triangle, while not absent, becomes less in height with the change to the lateral posture.

Marked dulness occurring posterolaterally at the base on both sides is commonly produced by *hydrothorax*, or a dropsical transudation into the pleura. However, the amount of fluid is rarely the same on the two sides. The upper limits of dulness vary also with posture, although the mobility is apt to be a little more noticeable than where there is inflammatory pleuritic exudation present.

A band of dulness at the base sharply passing into a large area of tympanitic sound above is significant of *hydropneumothorax* or *pyopneumothorax*. Here the mobility with changing of posture is most free, as evinced by the prompt changing of the boundaries of dulness due to the rapid gravitation of the serum or pus, as the case may be.

Finally, in rare cases, a dull note may be caused by an *intense pneumothorax*, the air distending the pleural sac to the utmost, thus abolishing elasticity and resonance completely, and by tumors of the pleura, the latter giving rise to irregularly situated and outlined areas of deadness.

The diagnostic value of *regional dulness* may be *summarized* by the following simple statements: (*a*) Apical dulness, in the great majority of instances, means tubercular consolidation, rarely pneumonitis, gangrene, or new growths; (*b*) dulness in the interscapular region indicates usually tuberculous bronchial enlargement or tuberculous infiltration of the posterior borders of the lungs, while on the left side it may mean aneurism of the descending aorta; (*c*) dulness over the lower lobes—that is, in the lower axillary, and especially in the infrascapular regions—points to pneumonic consolidation or pleural effusion most commonly, although it may, in rare cases, be produced by infarct, gangrene, and tumors, and when not extreme in quality hypostatic congestion, edema, or collapse (atelectasis) of the lung may be inferred.

B. *Exaggerated or Abnormally Clear Resonance; Hyperresonance; Vesiculotympanitic Resonance*

Increase in the clearness and fulness of vesicular resonance, generally or locally, indicates an increase in the amount of air in the lungs, or its presence in the pleural sac (*hyperresonant tone*). This sound has the quality of normal resonance, but the intensity is louder, the pitch lower, and the duration longer than in health, and the resistance to the percussing fingers is less apparent.

(1) **Slightly exaggerated resonance** is usually found over a *local* increase in the volume of air, as over a sound lobe adjacent to one that is consolidated, the healthy part performing extra work to compensate for the crippled area.

(2) **Moderately exaggerated resonance** is characteristic in healthy young children, with their thin and elastic chest walls. *Unilaterally*, it occurs in percussing over the overdistended, unaffected lung that is temporarily doing double labor because of consolidation or compression of the opposite lung. This functional enlargement of one lung is often called "vicarious" or acute *compensatory emphysema.*

In pathologic or permanent overdistention of both lungs in true *emphysema*, the exaggeration of resonance in the early stages of the disease is but moderate. In extreme anemia, also, probably on account of the lessening of the relative quantity of blood in the lungs, a certain intensification of the resonance may be elicited.

If, with the vesicular dilation, the tension is greatly increased, as in some cases of compensatory emphysema, the pitch may be slightly raised because of the dominance of that factor over the increase in size of the air-cells.

(3) **Hyperresonance.**—Abnormally marked clear, deep, and loud resonance is typically heard over the whole chest of one having well-developed *emphysema.* The emphysematous sound has been well designated the "bandbox note." Its quality approaches nearly to that of the tympanitic. It is accounted for by the double pathologic condition of increased volume of air from permanently dilated vesi-

cles and decreased tension because of the weakened elasticity of the vesicular substance, both causing a marked deepening of the pitch. Hyperresonance is also heard typically in *pneumothorax*, usually over one side.

(4) **Vesiculotympanitic Resonance.** SKODAIC RESONANCE.—Although essentially synonymous with the quality of hyperresonance, the sound first described by Skoda, and later designated vesiculotympany by the American clinician, Austin Flint, Sr., requires separate consideration because of its usage in these terms, and the fact that it occurs in special conditions with characteristics of a slightly *higher pitch* and sometimes more *tympanitic* attribute than the loud and grave note of hyperresonance.

The discrimination may be made in this wise: if the sound elicited is that of loud and low-pitched exaggerated resonance on both sides, or on the greater part of one side, bilateral enlargement of lung, as in senile emphysema, or unilateral diffuse or closed pneumothorax are inferred; but if, with increased intensity of note the pitch is at the same time higher than over an adjacent part on one side of the chest, particularly if the quality is almost tympanitic, the Skodaic resonance heard indicates a partial relaxation of the underlying vesicular tissue, as over an upper lobe when the lower is solidified in the second stage of pneumonitis, or just above the level of the fluid of a pleural effusion. In the latter case the Skodaic resonance is typically in evidence when the quantity of exudation is sufficient to fill about one-third, one-half, or even two-thirds of the pleural sac, so as to float the lung upward and diminish its volume and intravesicular tension without completely compressing it. In percussing from below upward posteriorly, the transition from the pleuritic dulness to a tympanitic hyperresonance at the level of the angle of the scapula, for example, is quite characteristic; and yet, as compared with the sound over the unaffected lung opposite, the pitch is a trifle higher, even taking into consideration the slight elevation of pitch from increased tension which the overacting normal lung in a state of compensatory emphysema is laboring under.

Vesiculotympany in the neighborhood of the heart may point to localized diminished pulmonary tension due to a *pericardial effusion*, or, less commonly, to the encroachment of a much-enlarged heart. Likewise, decreased tension of the lower borders of the lungs due to a very high position of the diaphragm from great abdominal enlargements may be demonstrated by the vesiculotympanitic note obtained.

C. *Tympanitic Sound and its Variations*

The tympanitic sound is not heard over normal lung tissue; therefore its presence always indicates disease of the structure directly beneath the percussing fingers, or quite adjacent to the region percussed.

The pathologic conditions giving rise to tympany are seldom of such size as to cause the pitch to be as low as that of the normal pulmonary resonance. Broadly speaking, the comparatively low-pitched and louder (although less grave and intense than the vesicular resonance) tympanitic sounds are produced by absolutely relaxed lung, and large pulmonary and pleural cavities, from whatever specific cause; the higher-pitched by small cavities filled with air, and small, circumscribed areas of dilated or relaxed vesicular tissue.

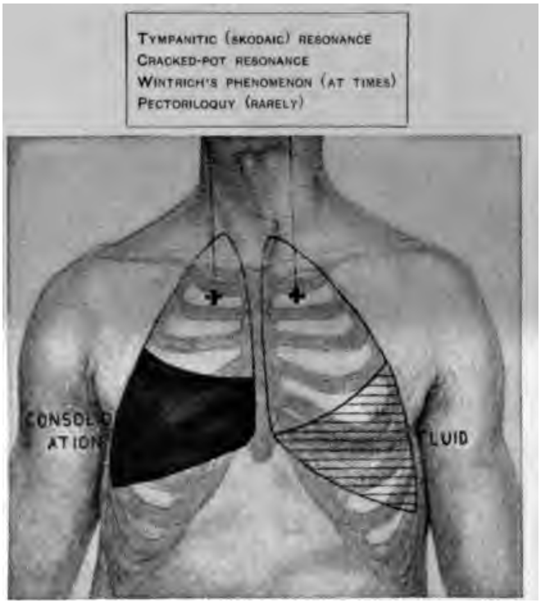

TYMPANITIC (SKODAIC) RESONANCE
CRACKED-POT RESONANCE
WINTRICH'S PHENOMENON (AT TIMES)
PECTORILOQUY (RARELY)
ONSOL
ATION
LUID

FIG. 27.—SHOWING CERTAIN PERCUSSION AND AUSCULTATORY FINDINGS ABOVE CONSOLIDATIONS OR EFFUSIONS. (Butler.)

(1) **Tympany of Relaxed Lung.**—As an occasional exception to the preceding general statement, a case of such marked emphysematous enlargement coupled with extreme lowering of pulmonary tension may be met in which the tympany exhibits very low pitch and pronounced loudness of tone all over the chest.

Tympany is to *complete retraction* of the lung what vesiculotympany is to partial relaxation. In the former condition the vesicles are in a state of *elastic equilibrium,* as when the lungs are removed from the body with their size reduced from the state of collapse and the absence of the intravesicular inspiratory tension.

When the cause of the relaxation resides within the lung, the tympany is said to be due to *immediate* relaxation; this occurs in the first (congestive) and third stages of lobar pneumonitis and in edema of the lungs, the diminished tension being the result of capillary engorgement (first stage), the presence of fluid and air in the vesicles (third stage), or of transuded fluid and air (edema). The tympany in these cases is slightly dull and of moderate pitch, according to the volume of air present. A small area of high-pitched tympanicity may be due to lax intervening air-containing tissue between a tubercular or bronchopneumonitic deposit and the surface.

Mediate relaxation of lung is produced by some extrapulmonary cause in the pleural sac, interfering with the expansion of the lung. The medium intervening to cause the retraction is most commonly a large pleuritic exudation. Elastic equilibrium of the lung manifested by tympany may also occur from large *pericardial effusions* or *tumors* in juxtaposition, from *subphrenic abscesses,* and from great abdominal distention as witnessed in general peritonitis.

On account of the diminished size of lung, its compression even in some cases, so that one percusses hardly more than a bunch of air-containing bronchial tubes, the tympany is invariably higher pitched than the normal resonance on the unaffected side, and also because of the muffling due to the relative preponderance of tissue minus air, and the dulling effect of the encroaching fluid.

The occurrence of *apical tympany* of high pitch, due to early tuberculosis, is sufficiently frequent and important practically to justify reiteration. That this sign may be misleading, relative dulness being attributed to the sound and really resonant apex, is evidently because any dulness to which a group of nodules might give rise is masked by the surrounding relaxed vesicles of the lung; hence the diagnostic value of the most careful technic that the true tympanitic quality with slightly lower and louder sound than over the healthy apex may be recognized.

(2) **Tympany over Pulmonary Cavities** (*Vomicæ; Caverns*).—Localized areas of tympanitic sound, more or less clear according to the conditions about to be pointed out, indicate usually air-spaces or cavities due to the destruction of lung tissue by *phthisis* (*tubercu-*

losis), *gangrene; abscess, actinomycosis*, or *circumscribed bronchial dilation* (*bronchiectasis*).

The conditions which make it possible to hear the tympanitic percussion sound over lung cavities, and which affect its clearness or intensity, especially its pitch, are the following: size, situation, communication, form, quantity of liquid present, thickness (rigidity) and smoothness of walls, condition of surrounding lung and pleura.

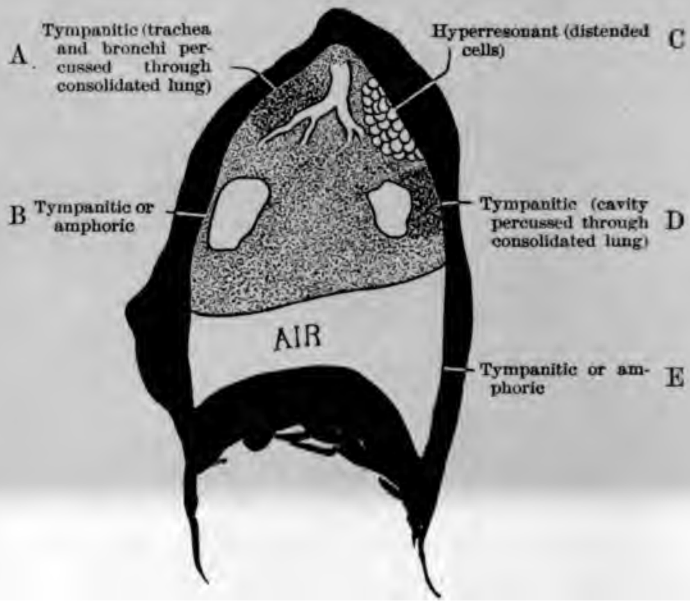

A Tympanitic (trachea and bronchi percussed through consolidated lung)
C Hyperresonant (distended cells)
B Tympanitic or amphoric
D Tympanitic (cavity percussed through consolidated lung)
AIR
E Tympanitic or amphoric

FIG. 28.—DIAGRAM SHOWING THE PHYSICAL CONDITIONS WHICH CAUSE HYPERRESONANCE AND TYMPANITIC OR AMPHORIC PERCUSSION SOUNDS. (Butler.)

As all of these conditions are interrelated, the audibility of the cavity sound will depend upon their combination rather than upon any single one. Other things being equal, however, to detect a cavity, its *size* must not be smaller than a walnut (1 to 1¼ in. in diameter), and for that size it must lie near the surface. Consequently, cavities of the apex are more easily discovered because necessarily superficially situated, with thin chest wall intervening; over deep-seated cavities, even when of considerable size, only the faintest tympanitic quality may be elicited, if at all; the apical region of the chest wall being naturally thinner than that over the lower lobes, covered as it is with thick dorsal muscles, therefore suffers more from the general emaciation associated with the presence of cavities.

The tympanitic note over a pulmonary cavern is rarely so clear or loud as intestinal tympany, as it is produced by the vibrations of a much smaller body of air.

The physical characteristics of tympanitic resonance are more distinct if the cavity communicates with a large bronchial tube open to the outer air, the sound reverberations being freer than when the cavity walls are closed (*open* and *closed tympanitic sound*). Besides, the hindrances to the development of a tympanitic sound, as thickened cavity walls or intervening normal lung tissue, weaken its distinctiveness more as the closed than as the open variety, since it lacks the additional vibration of the bronchotracheal column of air.

The influence of the *shape* of a cavity upon the tympanitic note will be described later under the consideration of the mutations of the pitch.

If the cavity is partly filled with liquid, as not infrequently happens temporarily, the tympanitic resonance becomes relatively deadened, and of course absolutely so if nearly or quite full.

Smooth, thin-walled cavities give forth a clearer tone of tympany than those having rough, thick walls. The capacity of the former to vibrate with their contained air is obvious.

More or less muffling or deadening of tympany may be caused by percussing a cavity covered with thickened lung or pleura. This *dull* or "boardy" *tympanitic* sound may become absolutely dull where the consolidation or pleural thickening increase markedly, and in some cases cavity has been revealed post mortem that during life was overlooked because of the great thickening between it and the chest wall preventing the penetration of the percussion vibrations.

The conditions causing changes in the pitch of the tympanitic sound over a cavity are notable.

(*a*) TENSION OF ITS WALLS.—Over a cavity with lax walls the tympanitic sound is clear and low in pitch, with a hollow or *cavernous* character. On the other hand, cavities with firm or rigid and tense walls produce a note which is described as *amphoric*, equally clear and almost metallic in resonance, but higher pitched than the other.

(*b*) WINTRICH'S CHANGE OF SOUND.—This phenomenon is a rise in pitch and augmented loudness of the tympanitic note when the patient opens the mouth, protrudes the tongue a little, and at the same time breathes as lightly as possible. The percussion strokes should not be too strong. When the mouth is closed the pitch is lower, and the sound less distinctly tympanitic; the pitch is lower still when the nostrils are closed. This simple Wintrich's change of sound occurs over those cavities that communicate freely with one of the bronchi.

The paradoxical combination of louder and clearer tympanitic sound with higher pitch is not actually a contradiction of the law of physics that, other things being equal, the lower the pitch the louder is the sound; but right here is the point of the matter, for the physical conditions are not equal during the percussion of a cavity when the mouth is opened and when it is closed. In percussing over a cavity communicating with the bronchotracheal column of air, the mouth being closed, the tympanitic note is comparatively dull and yet low in pitch, owing to the confined reflected sound-waves becoming waves of interference, and simultaneously slower waves. When the mouth is opened, however, since sound-waves in air travel longitudinally, at the instant of opening the percussion drives the waves out with accelerated velocity, crowding, as it were, a number of them with some friction through the oral orifice into the more quiescent outer air at the lips; the number of sound-waves per second received into the outer air is thus greater than the number sent out, and the pitch rises (Doeppler's Principle). Besides this, an important contributory factor in the elevation of the pitch on opening the mouth is the enlarging of the external orifice of the cavity, as it virtually becomes thereby, obeying the law that the larger the orifice the higher the pitch.

It remains to state that when the mouth is opened during percussion, the mouth-throat cavity acts as a resonator to the sound-waves expelled from the pathologic cavity, the resulting consonance giving greater fulness and intensity to the tympanicity.

In certain cases where the tissue around the cavity is markedly thickened, the merest trace of tympany only may be noticeable in percussing with the patient's mouth closed, while with the mouth open the unmistakable signs of cavity are discovered in a clear, tympanitic sound, louder and higher in tone, by this Wintrich's method.

The change of sound may be simulated by percussing over the larynx or trachea while opening and closing the mouth; also in tapping the cheeks similarly, or by blowing between the thumbs of the clasped hands—as for whistling—and then suddenly opening them.

Wintrich's change of sound may occur also in cases of *pneumothorax* in which there is a fistulous communication of the pleura and lung between the pleural sac and a free bronchus. Rarely, the phenomenon may be elicited over the upper lobes of the lungs in pneumonitis when the lower are consolidated, or when they are collapsed by a pleural effusion.

(c) INTERRUPTED WINTRICH'S CHANGE OF SOUND.—This is a change in the distinctness of this sign, or even in its occurrence at all, by changing the position of the body. That is, in one position—in sitting, for example—the bronchus leading to the cavity is open, and the sound tympanitic, while in the other—lying down—the bronchus dips beneath the surface of the liquid secretion, is thus closed, and the sound is either faintly tympanitic or dull; these manifestations may be reversed for the same postures. Such behavior, though rarely met with, when it is becomes positive evidence of the presence of a cavity.

(*d*) GERHARDT'S CHANGE OF SOUND.—This is another variety of altered pitch on changing the position of the patient, nor frequently met with, affording strong proof of the presence of cavity, however, when elicited. It is indicative of large cavities, oval or elliptical in shape, and partly filled with fluid the mobility of which with change of posture alters the form of the air-containing part of the cavity, its relation to the chest wall, and consequently its pitch. The latter is lower when the long diameter is horizontal—that is, when the cavity is of such a form and so situated that its long diameter is about parallel with the long axis of the body, the pitch is lower with the patient in the recumbent position, the fluid gravitating away from the chest wall; while when the long diameter is vertical, as in the sitting or standing posture, the sound becomes higher, deadened, tympanitic, because the fluid is now in closer contact with the chest wall; on the other hand, if the cavity's long diameter corresponds with the anteroposterior diameter of the thorax, the percussion note is lower in the upright position, while the column of air is shortened when the patient lies down, and the pitch rises.

Gerhardt's change of sound may take place over communicating as well as over closed cavities. Change in the tension of the cavity wall may also enter into the production of these changes of pitch.

(*e*) RESPIRATORY CHANGE OF SOUND (*Friedreich's*).—In this phenomenon the tympanitic sound over a cavity becomes higher in pitch at the height of a deep inspiration. It results from increase of tension of the cavity walls, the chest wall, and the adjacent lung substance, perhaps. In expiration the sound becomes deeper or it may disappear, especially during the act of coughing, because of collapse or compression of the cavity walls. The inspiratory rise in pitch may be partly due also to the simultaneous widening of the glottis.

(3) Williams's Tracheal Tone or "Resonance."—Closely allied in character to Wintrich's change of sound, the conditions which give rise to it are, however, different. It is the term applied to a tympanitic sound obtained in percussing over trachea or main bronchus (cylindrical cavity), the pitch being higher with the mouth open and lower and yet less loud with the mouth closed, provided the conduction of sound between the bronchotracheal column of air and the chest wall is facilitated by *retraction* or by *consolidation* of the intervening lung tissue. In the former condition, as from the shrunken lung of fibroid phthisis or the collapsed or compressed lung of a large pleuritic exudation, the bronchus is exposed by the retracted anterior border of the lung; in the latter, the bronchial tympany, with accompanying change of pitch, is brought out by the better conduction of sound through the hepatized lung of superior lobar pneumonitis (dulness with tympany), or of a large tuberculous consolidation of the apex; or, again, through the condensation of a lung by pleural effusion that does not separate it too much from the front of the chest.

This change of pitch, in opening and closing the mouth, is obtained normally in percussing over the larynx and trachea down to the bifurcation of the latter, at which point, and over the primary bronchi, it is lost, owing to the covering of vesicular lung substance; therefore, the detection of Williams's tracheal tone here signifies a pathologic condition causing either retraction or pneumonic infiltration of the lung usually.

The phenomenon is best heard, then, in percussing the first and second intercostal spaces near the sternum. The flexibility of the sternal ends of the ribs and of the costal cartilages favors the production of the sound.

That the tracheal resonance of Williams is found more often on the left side is due probably to the greater length of the left bronchus.

To *differentiate* Wintrich's change of sound from Williams's tracheal tone, the following considerations may be helpful: (1) Whether the visible contraction is marked and local, when it is likely caused by an apical cavity; absent or almost unilateral when it is a tracheal tone from hepatized lung or shrunken anterior border. (2) The strength of stroke required to elicit the sign; the change of pitch over a cavity (Wintrich's), is easily obtained with light percussion, while if a forcible stroke is necessary, bronchus (Williams's) is indicated. (3) Other signs of cavity are more evident when the change of sound is Wintrich's.

A change of sound similar to Williams's occurring over the upper sternum when percussed, where normally it is absent, indicates the presence of pathologic thickening or new growth between the trachea and manubrium. The cause may be a mediastinal fibro- or lymphosarcoma, an aneurism of the arch of the aorta, or a pericardial effusion displacing the heart upward.

(4) **Tympanitic Sound in Pneumothorax.**—This is rarely met with, and occurs in cases that are circumscribed and open; that is, practically a pleural cavity walled in by adhesions and communicating with the bronchial column of air through a fistulous opening in the pleura. Hence, also, Wintrich's change of sound may be elicited here. Should the expiratory current of air be prevented from escaping by the formation of a valvelike closure of the pleural opening, thus causing the tension to rise considerably in the interior because of the successions of inspiratory entrance of air while the pressure is sufficient, then the note becomes hyperresonant, as in the more common diffuse or closed pneumothorax, though not so loud, nor so grave in pitch.

D. *Amphoric or Metallic Resonance*

The *amphoric* or *metallic* note is fundamentally tympanitic, with added overtones giving an echolike quality and prolongation, or metallic clang. It is often higher in pitch than the ordinary tympanitic sound. While the latter ceases immediately after the percussion stroke, the overtones of this amphoric resonance, or metallic echo, die away more slowly. It is like the reverberating prolongation of the voice heard in speaking in a large, bare room or empty hall. It may be simulated by percussing or filliping the side of an empty, or nearly empty, jar or pitcher, a large, hollow india-rubber ball, or the cheeks with the mouth open.

Thus, the amphoric sound is heard over large, tense or rigid, and smooth-walled cavities of the lung, superficially situated, or over open pneumothorax. If produced by a tuberculous cavity, it means that the cavity is single or entire, and not subdivided by projections or partitions of pulmonary tissue that has not disintegrated; that it is distended with air; that it communicates more or less freely with a large bronchus; and that its size is probably not less than 2½ in. (6 cm.) in diameter in the direction in which the percussing stroke is applied. The marked emaciation usually present in these cases offers the minimum of resistance by the chest wall, and so facilitates the development of amphoric or metallic echo.

The modifications of pitch, as in Wintrich's change of sound, may also be elicited here; and, too, the variations due to cavities not spherical in shape, as the lower pitch while percussing in the direction of the long diameter (Gerhardt's change).

Bronchiectatic cavities do not manifest the amphoric resonance because there is less wasting, they are situated in the lower lobes away from the surface and where the thicker posterior wall of the thorax intervenes, and because they are too small in size.

Metallic sound is very distinctly heard over pneumothorax, provided the air contained in the pleura reaches a certain degree of tension (not too exaggerated). This condition may be inferred if heard over a large area of one side.

In order to appreciate amphoric resonance more distinctly, percussion with a hammer and pleximeter may be resorted to, with or without simultaneous auscultation. In combining the latter method, a clear, metallic note is brought out by placing the chest piece of the stethoscope near the pleximeter, which is then lightly struck with the hammer.

Coin or *rod-pleximeter percussion*, or the *coin-clinking test* (Gairdner), is most effective in discovering pneumothorax. It is practised thus: Two coins of sufficient size—say 25- or 50-cent pieces—one applied flat to the chest, the edge of the other used as a plexor, are used on the anterior surface by an assistant, percussing lightly, while the examiner auscults posteriorly, or *vice versa*, either with or without a stethoscope. Instead of coins the handle of a hammer plexor, or a rod of metal or pencil, may be used to strike upon the pleximeter, as recommended by Heubner. The presence of air in the pleural sac is indicated by a clear, ringing, chiming sound, bell-metal or anvil-like in character; the sound may be compared to the tinkle of a small bell (*bell-tympany*) or the effect of striking an anvil with a tiny hammer. Comparing the two sides of the chest, the absence of the sign is conspicuous over the lung on the unaffected side, and over the lung on the affected side, from which the air-space in the pleura may thus be sharply delimited.

Biermer's Change of Sound.—As pneumothorax is often accompanied or followed by liquid effusion into the pleural cavity, causing pyopneumothorax, for instance, the region over which amphoric resonance may be elicited becomes gradually diminished in extent, so that the amount of air remaining may give rise only to a tympanitic sound on percussion, the metallic overtones disappearing entirely.

On *changing the posture* of patients suffering with pyo- or sero-

(hydro-) pneumothorax, the pitch of the note changes, being lower or higher according as the long diameter of the cavity is rendered longer or shorter, respectively, as the fluid gravitates over the diaphragm or upon the back, the patient sitting or lying down (so Biermer). On the other hand, however, many clinicians have noted the change to be reversed in some cases; that is, the diameter may be shortened while the patient is erect and lengthened while recumbent, with corresponding change from higher to lower, respectively. But, whatever the given conditions of alteration of the pitch as affected by posture, the fact remains that, as with Gerhardt's change of sound in cavities of the lung, so Biermer's change indicates the presence in the pleural sac of both air and freely movable fluid.

E. *Cracked-pot Sound* (*Bruit de pot fêlé*)

The *cracked-pot percussion sound* is frequently heard in connection with amphoric resonance. As its name implies, this sound resembles that produced by percussing a cracked jar or metal vessel, and may be imitated by loosely clasping the hands at right angles to each other, and then sharply striking them (the back of one) across the knee. The concussion produces a sound which closely resembles the chinking of coin held in the hollow of the hands—a rattling, sometimes peculiar hissing, metallic clinking, tympanitic sound. The cracked-pot characteristic may also be imitated by percussing a pleximeter so lightly applied to the skin that beneath it is left a small space containing air. In both experiments the sound is caused by the sudden discharge of air from between the palms, or the skin and pleximeter, as the case may be.

This is virtually what happens when the cracked-pot sound is elicited over the chest by a forcible percussion stroke: it means usually that the air in a pulmonary cavity communicating with a bronchus is sharply expelled through the narrow, slitlike opening at the glottis.

An abundance of crisp hair on the chest anteriorly may so interfere with the snug application of a pleximeter that an air-space, as just previously pointed out, may cause the cracked-pot sound to be simulated when the pleximeter is struck unless the hair is well moistened first, so that it will lie close and flat on the surface. The sound may occur normally, also, when percussing the chest of a screaming child, though not always necessarily so, on account of the very thin, yielding thoracic walls of infants. Very rarely is it heard in healthy adults.

Pathologically, it is produced:

(*a*) Over *large lung cavities*, superficially situated, especially at the apex, when they communicate directly with a bronchus by means of a small opening, and the overlying chest emaciated to thinness and elasticity. The patient's mouth should be open, and percussion made during expiration, so that the sudden expulsion of the cavity air through the opening may have a free egress to the outer air, otherwise the hissing, clinking sound distinctive of cavity may not respond. It is almost invariably well heard in the infraclavicular regions. It disappears at times during the course of a case that is repeatedly examined, owing to closure of the bronchial opening and the filling of the cavern with exudate, to return again after the substance has been expectorated by a spell of coughing. Another condition for the production of the cracked-pot sound, which indeed is indicated by the occurrence of the latter, is the thin, yielding wall of the cavity itself, whether composed of merely slightly thickened pleura on its parietal aspect or of consolidation of surrounding lung.

The percussing finger should pause a moment after striking, for if lifted too soon the tympanitic sound significant of cavity may not be elicited, as it is apt to be subdued at the first instant by the hiss of the immediate rush of air from the cavity into the bronchus.

The presence of a moderate amount of liquid in the vomica may be inferred by a commingled rattling and trembling sound—the *moist cracked-pot sound.*

(*b*) In certain cases of *pneumothorax,* usually *circumscribed,* with *patulous pleural opening* in direct communication with a bronchus. In this condition the cracked-pot sound is found over the lower half of one side of the chest, in contradistinction to that of pulmonary cavities, where it is discovered over a smaller area in the upper half, and more often anteriorly than laterally or posteriorly.

(*c*) Similarly, in *thoracic fistula,* as when an opening has been made into the pleural sac for the evacuation of pus of an empyema, or when air has entered into the pleural cavity through stab or gunshot wounds, whereby percussion in the immediate neighborhood of the orifice elicits the cracked-pot note, which is also absent when the orifice is artificially closed.

(*d*) Over *pneumonitic lung.* This occurs more commonly during the congestive stage, or while there is but partial consolidation, in which condition, it will be remembered, the accompanying immediate relaxation of lung causes a tympanitic percussion sound. However, the phenomenon may be elicited not rarely even over the hepatized portion of lung (especially over the upper lobes anteriorly), where its production must necessarily be due to the sudden expulsion of air from a large bronchus leading to the part affected. In some cases both causes may exist simultaneously to produce the *bruit de pot fêlé.*

(*e*) Finally, *retraction* or *compression* of the *lung* above a *pleuritic effusion* favors the development of cracked-pot sound by affording the condition of a sharp rush of air from the bronchi with strong percussion strokes.

In view of the variety of pathologic conditions, though small, which may operate to give rise to cracked-pot sound, the warning is obvious—to avoid attributing its presence to any one of them. Nevertheless, its detection in any patient evidently phthisical is rather clearly presumptive of pulmonary cavity, the other causes occurring much less frequently and characteristically.

Sternal Percussion.—An increased resonance over the sternum may indicate the presence of a cavity in the adjacent lung. When the resonance over the sternum persists as the patient reclines, but vanishes when he lies on one side, this can be suggestive of hydrothorax or pyopneumothorax. Banti was the first to call attention to the retrosternal dulness observed in case of pleurisy with effusion, which aids in differentiating it from pneumonia.

F. *The Lung Reflex*

This term has been applied by Abrams to the temporary localized emphysema or expansion of lung, in response to an irritant applied to the skin of the chest, such as mustard, so that the percussion sound becomes for a little while clearer in that area. But the effect of prolonged percussion in itself is similar, the continued repetition of strong percussion strokes giving rise to a development of increased resonance and volume of sound in many cases. Therefore, one must not be surprised or perplexed to find in prolonged examinations or demonstrations of small areas of dulness, as in tuberculosis, that the later percussion blows may elicit more and more of the surrounding resonant quality, due to this reflex localized expansion of the air vesicles, and correspondingly less dulness or muffling.

To *summarize*:

(1) *Vesicular resonance* is obtained over normal lung tissue.

(2) *Tympanitic resonance* is heard normally over the lower portion of the left side anteriorly, in Traube's semilunar space (lung and stomach). Elsewhere on the chest, tympanitic sound is pathologic.

CHAPTER VI

AUSCULTATION

Definition.—*Auscultation* is the act of listening to the sounds produced within the body in health and disease. Comprehensively, it embraces the physiologic and pathologic sounds of the lungs, heart, and gastro-intestinal tract; here it refers to those produced in the act of breathing, and, of lesser importance, to those set up by voice vibrations transmitted to the lungs and chest wall.

Although we may listen and take note of the sounds of coughing, wheezy asthmatic breathing, or thoracic splashing sounds at a certain distance from the patient, auscultation proper means the direct or mediate application of the ear to the surface of the chest for the purpose of ascertaining the significance of the various sounds produced within. Hence, as suggestively described by A. L. Loomis, auscultation is "a kind of eavesdropping"—listening with bent head and applied ear while all the time the conditions are unseen.

Historical Note.—That Hippocrates should have observed and described some of the phenomena of auscultation is not to be wondered at so much as that, for centuries afterward, so few, scattered, and obscure references to auscultation should have been made. Thus, he described and named the succussion or splashing sound heard in pyopneumothorax, and, from certain passages in his works, was undoubtedly acquainted with the creaking friction sound of pleuritis and the rattling sounds of catarrhal (bronchial?) affections, although evidently not impressed with their real significance.

It was not until after percussion had been utilized in a limited way for fifty years that Laennec (1781–1826), the inventor of the stethoscope, first practised auscultation. This was in 1816. Three years of diligent application and observation enabled him to become so proficient in the method that he had established the diagnostic value of most of the auscultatory signs, and forthwith wrote his classic work, in 1819, the "Traité de l'Auscultation médiate et des Maladies des Poumons et du Cœur." Skoda's critical analysis of

Laennec's results, and his own contributions of additional data, especially in demonstrating the physical causes for each of the auscultatory signs discovered, has given us an exactitude of information and a simplicity and practicality of classification regarding these phenomena that has hardly been improved upon since.

In later years, much development of skill in this method, and of precision in the inferences derived from the physical signs elicited, as a result of able and frequent and painstaking clinical and pathologic investigation, gives auscultation the most prominent, productive, and promising position as a single method of physical examination among the others in the majority of cases. It has the advantage that percussion lacks, namely, comparative easiness of technic. Acuity and reliability of hearing and concentration of attention are as necessary in auscultation as in percussion, often even more so, because of the mingling of the respiratory and cardiac sounds; nevertheless, perseverance usually begets a grasp and mastery of both methods often so soon and suddenly as to be surprising.

METHODS

Auscultation, like percussion, may be conducted in two ways: *immediately* or *mediately*. In the former, the ear is applied directly to the chest wall, with nothing but a napkin, thin, soft (unstarched), towel, or handkerchief intervening; in the latter, or indirectly, help is derived by the use of an interposed, tubular, conducting instrument —the **stethoscope.**

Both methods are used, and are useful, but ordinarily the immediate method is, perhaps, more serviceable, which is quite the opposite in examining the heart, where a stethoscope is essential in determining the various valve sounds and murmurs. In general, it may be said that immediate auscultation gives us a better idea of the condition of a relatively large area of the lung, and sometimes of deep-seated consolidations and cavities, than does mediate auscultation. On the other hand, for circumscribed, detailed work and the more superficial pathologic areas, the aid of a stethoscope should be resorted to.

The subjoined advantages and disadvantages of the two methods may be enumerated, to serve as a guide in judgment as to which one, or whether both, should be employed in any particular case. The principal ***advantages of immediate auscultation*** are, that (*a*) it is an easy and ready method of application to obtain a broad survey of the physical state of the lungs, and yet a fairly precise knowledge of not too small areas, while where rapidity of examination is required, as in emergencies and in states of exhaustion or grave illness, in which the patient is unable to sit up longer than a few moments, especially to have the back auscultated, it is almost indispensable; (*b*) the purity of the respiratory sounds is better preserved; (*c*) slight and superficial as well as deep changes of sound may be appreciated more distinctly with the ear placed closely. In some cases it has been my experience that certain râles and rustling pleuritic friction sounds, pathologic breath sounds even, as well as certain cardiac murmurs and the aneurismal bruit, were better recognized without than with the stethoscope; (*d*) palpatory sensitiveness of the applied ear helps to realize the character of the chest movements, feel friction thrills, and discriminate vocal vibrations. The disadvantages are counterbalanced by the following dominant

Advantages of Stethoscopic Auscultation.—(*a*) Accuracy of localization, enabling one to listen to sounds produced in a very small, limited space; (*b*) intensification of the sounds from any given circumscribed area; (*c*) where it is impracticable, for anatomic reasons, to apply the ear, as in the supraclavicular, axillary, and sometimes the suprascapular regions; (*d*) whenever, in using the unaided ear, the posture necessary to be assumed is constrained, inconvenient, or awkward; (*e*) sometimes for reasons of delicacy, in examining females, who may be sensitive in having the head applied to the breast region; (*f*) to avoid being soiled or contaminated by unclean clothing or bedding, and by parasites, or infected by contagious disease; (*g*) excludes external sounds (with the binaural stethoscope).

Among the **disadvantages** in the use of the stethoscope are these: (*a*) It modifies the true quality of the chest sounds by the adventitious roaring and other sounds due to the construction and materials of the instrument; (*b*) it intensifies such interfering sounds as may be caused by the rubbing of hair within the chest piece, or of adjacent garments during the movements of respiration; (*c*) it may require more time to examine the lungs than may be necessary or advisable; (*d*) babes are often too restless and tender to pressure, even slight, and children are frequently alarmed and irreconcilable at the sight of a stethoscope; (*e*) in very thin and emaciated persons, the circumference of the chest piece does not fit closely throughout, as the surface is too irregular on account of the narrow elevations and depressions of the ribs and intercostal spaces, thus admitting extrane-

ous sounds; (*f*) a trifling and yet practical objection is that the stethoscope is not always conveniently at hand.

The truth is that both methods should be learned, so that either may be adapted according to the circumstances and indications, attention being given first to the direct or immediate method of auscultation, and then to the mediate for special cases requiring detailed examination, as for tuberculosis, where the Bowles stethoscope is almost essential.

The Stethoscope and its Selection.—Stethoscopes are of two types and classes of adaptability and construction, the single or *monaural* and double or *binaural*, the solid or stiff, and the flexible. The stethoscope invented by Laennec consisted of one piece—a crude cylinder of wood. The simple stethoscopes are now made hollow, of hard rubber throughout, or of bell-metal tube and chest piece, or of bell-metal tube and hard-rubber chest and ear pieces, or of wood only. The tube is about 4½ to 6 in. long. The conical or bell-shaped thoracic end of the monaural stethoscope is preferably made of hard rubber, and but ¾ in. in diameter, and should not exceed 1¼ in. The hard-rubber ear piece should be large enough to cover the whole concha, and slightly concave—one that is 2½ to 2¾ in. in diameter is about right—and is usually detachable from the tube by a screw thread, for convenience of carrying in the pocket, as in the Hawksley, of London, stethoscope. The importance of a well-fitting ear plate has been emphasized by Walshe to be "as necessary . . . as to try on a new hat."

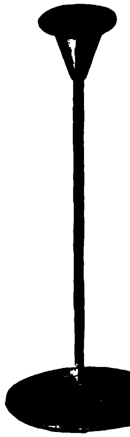


Fig. 29. - Hawksley's Single Stethoscope (two pieces). (Musser.)

While the SINGLE STETHOSCOPE has been used by many able clinicians and practitioners with satisfactory results in the localization and determination of the character of sounds; while it is handy and, for one whose hearing in one ear is deficient or lost, is quite sufficient, there are certain *disadvantages*, however, that should be pointed out here. In the first place, it does not shut out the external sounds free to enter the other ear; and, when the latter is held closed by a finger, roaring sounds are apt to be caused that may be equally confusing. Secondly, a common objection is pressure with the head, the weight of which is not as appreciable by the examiner as by the skin of the patient, to whom the almost invariable pressure marks represent unpleasant, if not painful, sensations. This discomfort I have seen augmented by the awkwardness which seems unavoidable, especially by beginners, of having the chest piece slip or tilt a little, thus digging more deeply into the skin on a part of its circumference, all because the auscultator must needs look away from the region of application of the stethoscope, and because his head is comparatively insensitive to the degree of pressure exerted or the accuracy of placing of the bell end.

The BINAURAL STETHOSCOPE, for reasons soon to be given, is now much more generally used than the single one, although relatively more often in auscultating the heart. A large variety of double stethoscopes has been devised since Dr. Cammann, of New York, in 1840, invented the first one, composed partly of metal and partly of flexible tubing, with hard-rubber or bone or ivory conical or cruller-shaped ear pieces attached to the metal tubes, and a bell-shaped hard-rubber chest piece to join the flexible ends. The auricular ends are usually held to the meatuses by a rubber band around the far ends of the metal tubes, or, more preferably, by either a curved flat steel, or coiled wire spring. A rubber band, naturally or because of accidental wettings, is more perishable as regard both its elasticity and continuity; it also frequently causes painful pressure in the ears when stretched to or near its utmost. This leads to the caution, that in choosing an instrument one should never be selected unless its adjustment to the ears is absolutely comfortable both as to shape of the ear cones and strength of spring. On the other hand, as in those with narrow heads, special care must be exercised to avoid a spring so weak and loose that the proper conduction of thoracic sounds to the ears is not annulled or interfered with.

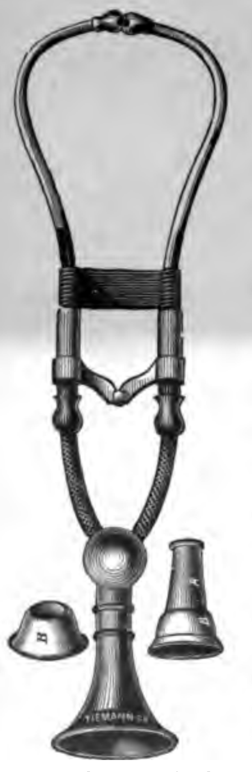


FIG. 30.—CAMMANN'S DOUBLE STETHOSCOPE AND SNELLING'S RUBBER BELL. (Musser.)

The *ear pieces* should curve so as to point in the direction of the ear canal—that is, downward and forward. Spheroidal ear pieces are less likely to press too deeply and uncomfortably into the ear canal than conical ones. The tubes need not be larger in caliber than the entrance of the auditory canal. The rubber tubing which joins the metallic tubes to the thoracic piece should be as soft and flexible as is consistent with purity and durability of material, and should be of sufficient length so as to allow the examiner to change the position of the chest piece frequently without having to move his head or body each time, as must be done with the stiff woven tubing, and so that the stethoscope may be conveniently folded for the pocket.

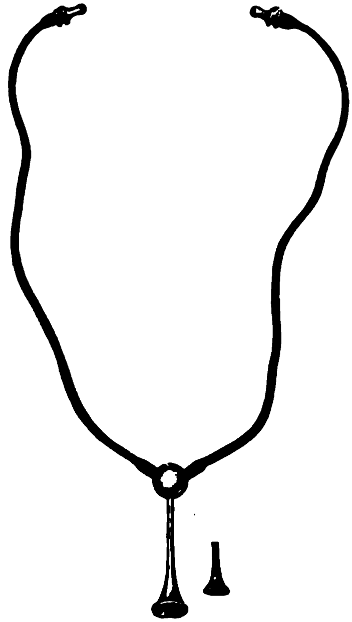


Fig. 31.—Simple Form of Sansom's Binaural Stethoscope. (Tyson.)

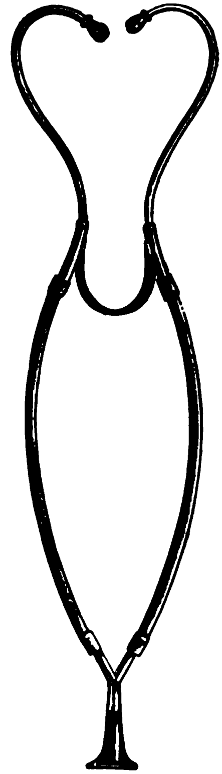


Fig. 32.—Binaural Stethoscope. (Musser.)

Jointed stethoscopes, however, whether of the hinged spring variety that fold longitudinally or vertically by the metallic tubing near the spring, or that are detachable where the metallic and metallic-tipped rubber or woven tubings meet, should be avoided. They annoy, and pervert and confuse the characteristic sounds of the chest by occasional squeaks and metallic creaks, intensified by the conductivity of the instrument; the smooth metallic junctures may also slip apart with a harsh shock to the ears and a disagreeable break in the auscultation.

The *chest piece* is made of hard rubber or wood, and in order that it may easily be placed in the intercostal spaces, should not exceed ⅞ in. in diameter. Ordinarily the chest piece should not be so thick-walled as to make its interior caliber at the upper half as small as in some makes of instrument, particularly if the bell is more than 1 in. in length, but should maintain a rather free opening to where the rubber tubes join it, so that the partial partition between them may be seen within the aperture.

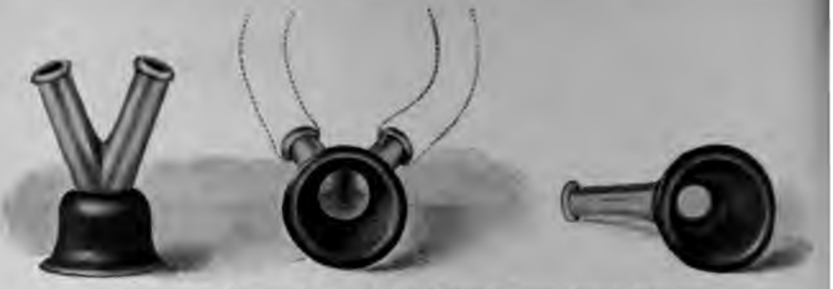


FIG. 33.—AUTHOR'S BINAURAL STETHOSCOPE CHEST PIECE.

When a larger thoracic piece is desirable, and one that will lie in close to the skin regardless of elevations and depressions, a soft rubber bell may be attached, as in the very satisfactory Valentine stethoscope, which conducts and augments the sounds remarkably well. The efficiency of this instrument depends partly upon the simplicity of construction and uniformity of material (mostly of hard rubber), and partly on account of the smooth finish of the interior of the tubing and the absence of unnecessary curves in the tubes connected with the ear pieces.

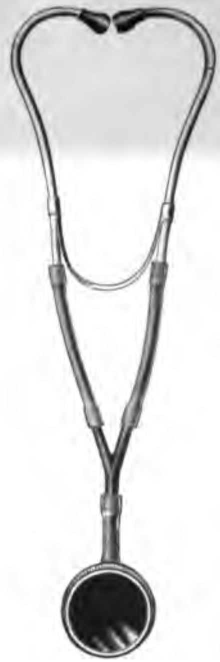


FIG. 34.—BOWLES' STETHOSCOPE. (Tyson.)

The BOWLES STETHOSCOPE deserves special notice as the most recent and adaptable sound intensifier in use. As shown by the illustration, it differs essentially from other binaural stethoscopes in the chest piece. This is somewhat like a telephone receiver, consisting of a shallow cup of steel, over the mouth of which is fitted a diaphragm of hard rubber, thin metal, or animal membrane, about 2 in.[1] in diameter, held in place by a steel ring. The disk prevents the projection of the chest tissues into the cup, and may also serve partly as a resonator, although this is denied by some clinicians. For bedside instruction, the Bowles stethoscope may have 2, 4, 6, or even 12 pairs of ear pieces joined to it, so that as many students may listen simultaneously with the demonstrator, and thus save the time of the examiners and the strength and patience of the patient. The transmission of thoracic sounds through so much tubing of the multiple Bowles stethoscope diminishes their distinctness as compared with their loudness when heard with the single instrument, but is about as effective as with the ordinary double stethoscope.

The *advantages* of the binaural stethoscope over the monaural are, briefly, greater clearness and intensification of sounds, directly because of the nature of the instrument, indirectly because with it external sounds are excluded more easily. Again, one sees precisely where the chest piece is placed while listening, that it does not slip, and feels sensitively with the holding finger and thumb that it does not press uncomfortably or unevenly, or rub against hair or clothing. And then, for those whose hearing is defective, the double, the Bowles stethoscope especially is very helpful. Also, with the latter one may quite often hear distinctly the pulmonary and cardiac sounds through several not too thick garments, as where rapid or emergency work may be required. And again, with the Bowles, the larger chest piece enables the examiner to auscultate almost as large a lung area as with the unaided ear. Finally, this same variety of stethoscope is an admirable safeguard and convenience in examining the posterior regions of the lungs in patients who may be too ill or exhausted, as from pneumonitis, to be moved to a sitting or even a side posture; for, by simply pressing down the bedclothes, the flat chest piece may easily be applied to the back and axillæ.

A binaural stethoscope may have these disadvantages, namely: to learners in particular, until accustomed to its use, the accompanying humming sound, especially if *slight*, tends to confuse or subordinate the true respiratory sounds, unless loudly manifest; but the rhythmic character of the functional sounds are soon readily differentiated from the instrumental hum. The larger size of the Bowles chest piece prevents its being used satisfactorily in the supraclavicular spaces, and between the ribs in emaciated subjects.

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[1] There is also a useful 1¼ in. disk.

The PHONENDOSCOPE, devised by the Italians Bazzi and Bianchi, though similar in the construction of its thoracic end, has no advantage over the ordinary stethoscope for general auscultation, and, on the other hand, is less convenient to carry and use. It consists of a metallic drum similar to but deeper than that of the Bowles stethoscope, and a little larger in diameter, to the under side of which two hard-rubber disks are attached, an inner for direct application to the chest, and an outer, which may be screwed down almost in contact with the other [illegible]ns of a retaining metallic, milled-[illegible]m. In the center of the outer disk is [illegible]ing, into which may be screwed a steel [illegible]bout 2 in. long and ⅛ in. in diameter, [illegible]he outer extremity of which a hard-rubber button is fixed, about ½ in. in diameter. The inner disk is applied when large areas [illegible] to be examined, more particularly of the [illegible]ungs, while the outer disk and buttoned stem are used when very small areas, especially the valves of the heart, are to be auscultated, or in the practise of auscultatory percussion. When not in use the stem is held by two small rings on the top of the drum, where also the soft-rubber conducting tubes, with their ear pieces, are attached. The phonendoscope is indeed a sensitive instrument, slight alterations in the breath sounds, mucous clicks, friction rustles, and murmurs being intensified to a more distinct audibility, and the changes of tone and pitch in approaching the borders of organs being mapped out by auscultatory percussion, or merely scratching the skin near the stem button as it is moved accordingly, are especially helpful in accurate physical diagnosis.

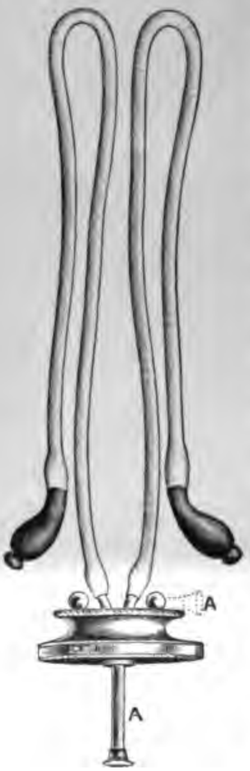


FIG. 35.—PHONENDOSCOPE: A, metallic rod.

The DIFFERENTIAL STETHOSCOPE, which is practically a divided binaural instrument, with two separate soft, flexible tubes attached respectively to a chest piece for each ear to receive sounds through, may serve a valuable amount of information in comparative work. Thus, it may be used to determine differences in time or rhythm of abnormal respiratory sounds over different localities simultaneously, or in the quality or loudness, synchronousness, or transmissibility of cardiac murmurs.

After all that has been said, the student should choose that stethoscope which best evidences good augmenting and transmitting capacity, simplicity and durability of make, comfort, and convenience in handling, and then *stick* to that until he is thoroughly accustomed to its individual peculiarities in conveying auditory impressions under various circumstances and in the varying conditions.

Technic of Auscultation.—*The patient* should be in a quiet room, so that the examiner may not be interrupted and handicapped by noises and talking. Both persons should assume easy, comfortable, and unconstrained positions. If possible, the patient should stand or sit, the arms hanging loosely, as for inspection, a blanket being thrown over the shoulders to avoid any chill. The attitude should also be symmetrical, any lateral or twisted posture of the body, as by supporting one hand, or resting on one foot, or leaning, causing unequal breathing movements on the two sides. In examining the posterior surface the patient bends the body slightly forward, folding the arms in front of the chest, as for percussion. In direct auscultation, the surface to be auscultated must have nothing between it and the observer's ear but a thin, soft towel, handkerchief, or light-weight undershirt, while for stethoscopic work the skin must be bared, in women as well as men, although in some cases determination and tact of manner and speech may be necessary. In those who are so ill that recumbency is imperative, after examining the anterior regions of the chest with the patient lying flat, he may be turned gently to either side, care being taken not to exhaust him by too lengthy an examination, but reserving the less essential regions for subsequent investigation. Then the back may be auscultated more conveniently by having the night-dress open there by a long slit from the neck down.

As it is necessary that both sides of the chest should be able to move with equal freedom, constricting clothing, corsets, braces, and the like should be released or removed; and the fallacious habit of auscultating one side and then the other, in front, while the patient alternately holds open and aside the intervening garments, must particularly be avoided, so as to prevent the imperfect and uneven results of muscle-sound interference and altered respiratory movement on the two sides due to muscular constraint.

At the *commencement of auscultation* it is usually advisable to listen to the breathing sounds while the patient respires as he is ordinarily accustomed or inclined to do. Some persons become nervously confused and puzzled when requested to breathe naturally; others require absolute and explicit guidance and directions, and coordinate their respiratory variations of movement quite satisfactorily. In all cases, a natural ease and freedom of expansion, with the mouth closed, should be aimed for. The respirations should be deep, gradual, and regular, and not violent, sharp or sudden, and jerky or rapid. Grunting, moaning, and moist clicking or rattling sounds in the throat should, as much as possible during auscultation, be suspended by instructions to the patient, or be allowed for as to their origin. We may aid the patient by performing the respiratory act properly ourselves, and then having him imitate us as closely as possible. Asking the patient to sigh helps to elicit a satisfactory breath sound; coughing may do likewise, the few inspirations immediately following being necessarily full.

To insure placing the ear exactly over the spot to be auscultated, it is well to put the tip of the index finger at the point desired, and then apply the ear so that the auditory opening corresponds to the place.

The examination should be conducted *systematically* and *comparatively*: we first auscultate the apices, then the lower regions anteriorly, then the lateral and posterior aspects of the chest, always comparing corresponding parts of the chest on the two sides. Often it is necessary to contrast different regions on the same side. No auscultation is complete unless the entire chest is examined carefully and critically, as evidences of disease may be discovered where such symptoms as pain may be absent. It is better not to linger too long over one spot, so as to avoid confusion of the ear by too marked an auditory impression, but rather to frequently alternate between symmetrical regions so as to note slight differences of sound that may have any pathologic significance. It is well also to get accustomed to using both ears, should any exigencies in regard to the disability of an inconveniently postured patient or on the part of one of the examiner's ears arise.

The Use of the Stethoscope.—Next to the selection of a suitable stethoscope is its proper application in practise. With the binaural the student can readily become accustomed to its intrinsic characteristics, and the normal breath as well as heart sounds, by applying the chest end to his own bared skin before using it upon that of his fellows and obliging friends, and then upon hospital attendants, convalescents, and patients.

The essential thing about the act is really mental rather than mechanical; that is, *concentration of attention* must be trained so that one learns to hear only those functional sounds to which the perception may be directed. In listening to the breath sounds, for example, the heart sounds are also heard with more or less distinctness, according to the location auscultated; but while the ear naturally receives the vibrations, the mind must hear the respiratory murmur alone, and deliberately disregard all other sounds for the moment, whether organic or external to the body. To hear the breath sounds merely is not difficult ordinarily, but to discriminate them from the extraneous and cardiac sounds, as one wills, requires training to attain, just as one may attend to the words spoken by another in conversation, or to the ticking of a clock, although the sound vibrations of both are being transmitted to the auditory apparatus.

The points of disadvantage of the single and binaural stethoscopes already enumerated may indicate some of the errors to be avoided in their use. Thus, care must be exercised that painful pressure is not produced by the head in using the monaural instrument, and that it does not tilt or slip, and so cause part of the chest-piece circumference to be without contact, or to prematurely change its location.

With the double stethoscope one sees the position of the chest end, and with the thumb and finger may press with just sufficient firmness to secure perfect, comfortable, and stable apposition, at the same time resting the little finger on the chest, if necessary. Patients who are standing or sitting while under examination, and who may be easily inclined to shrink away under the pressure of a stethoscope, should be supported by the examiner's free hand placed around the back, for instance, when auscultating the front of the chest.

Not only must one learn to disregard unavoidable *noises* from the street or building, or even in the room where the auscultation is done —although in the latter quiet should be insisted on—but also those extraneous sounds produced by the manipulations of the stethoscope, and especially the rubbing sounds occasionally caused by hair on the chest, or by an adjacent garment. Unless carefully recognized by moving the chest piece over the skin and noting the results, pleural or pericardial friction sounds may wrongly be inferred. A harsh, very dry, and hairy skin may be oiled so as to prevent any crepitating sounds of a deceptive character, or a soft-rubber cap may be put over the chest piece.

Clicking, snapping, or crackling sounds, due to slight motion of metallic hinged or adjusted parts, the vibrations of a spring, or the movements of the fingers holding the chest piece, may resemble abnormal adventitious sounds from within the thorax unless accounted for by close observation. Movements of the examiner's head while auscultating may result likewise. To avoid finger-friction sounds the chest end should be grasped so firmly that there may be no slipping. Kinking of the rubber-tube connections, or their contact with the skin or clothing, should be guarded against also.

It is important that the *ear pieces* point upward, as the metallic tubing is held horizontally before placing the stethoscope to the ears, as, if they are introduced in the downward pointing direction, their openings press against the wall of the ear canal instead of being directed axially, and thus humming sounds interfere with those from the thorax.

Finally, the importance of having the patient assume an easy, relaxed position during stethoscopic auscultation is seen in view of the fact that the sounds of muscular contraction are more readily perceived than when the ear is directly applied to the chest, and that they may be mistaken for râles of the "distant" or "indeterminate" type, sometimes referable to obscure or subacute pleural or pulmonary conditions. The tension of chest muscles is more noticeable during inspiration than expiration (over the pectorals in front and the trapezius and latissimus behind), especially in those who endeavor nervously to maintain an erect and favorable position.

Muscle sounds have a low-pitched, continuous roaring quality, or consist of several short, dull, higher-pitched rumbles rapidly succeeding each other. They are analogous to the sounds produced by strong contractions of the masseter muscle while the teeth are clenched; if at the same time the ears are held closed with the fingers, one hears typical muscle sounds.

The adventitious sounds or râles associated with pulmonary diseases may be distinguished from these muscle sounds by their more bubbling or crackling character, their clearer and more sharply defined quality, and shorter duration.

AUSCULTATORY PHENOMENA

Objects.—Auscultation is practised for the purpose of studying principally three classes of physical signs: (1) The ***respiratory murmurs***, or *breath sounds*; (2) *râles* and *friction sounds*—superadded abnormal sounds; (3) *vocal resonance*, or the sounds of the voice; transmitted cough and heart sounds may be included here.

THE SOUNDS OF NORMAL RESPIRATION

In listening over the normal chest we detect two types of *respiratory murmur*: (*a*) the *vesicular*; (*b*) the *bronchial*. A third variety of breath sound is heard normally which consists of a combination of these two types, and is therefore called *bronchovesicular breathing*—the "indeterminate" murmur of European writers.

These types of breath sounds are recognized, however, not by a mere naming of either as it may be found at a particular designated spot, but by an attentive, painstaking, critical examination and analysis of the attributes or elements which give them their special characteristics. This pertains to both the *inspiratory* and *expiratory* sounds. Thus, we learn to distinguish each variety of respiratory murmur by (as in percussion) the *quality*, *audibility* or *loudness*, *pitch*, *duration*, and the *relative length* of *inspiration* to *expiration*, or *rhythm*.

(1) **Bronchial Breathing.**—This type of breath sound is described first because it is easy to recognize, and, although not heard over normal lung parenchyma, is nevertheless the most commonly met with where the vesicular breathing is abolished, especially in pneumonitis and tuberculosis. Normally, it is heard, however, if one places the stethoscope against the trachea, just above the suprasternal notch, or over the larynx, or over either of the primary bronchi near their union with the trachea, at the upper portion of the sternum. The sound of bronchial breathing is harsh, blowing, tubular in quality. It is loud, high in pitch both during inspiration and expiration, depending upon the strength and rapidity of the breathings, though the expiratory sound is frequently higher pitched than the inspiratory, and each commences and ends with a sustained intensity and pitch. Both sounds may be equal in duration, but more often expiration is heard a little longer than inspiration. There is a slight break or pause between them, probably due to the fact that the inspiratory sound does not continue quite as long as the inspiratory act.

Skoda very well described the quality of the sound of bronchial breathing as corresponding closely to that produced by putting the mouth in the position of pronouncing the German "ch" or the rough aspirate "h," and then breathing deeply and regularly. It may be imitated also by blowing through a tube, as a single stethoscope of good caliber. The physical attributes of intensity, pitch, and duration of the bronchial breathing sound are characteristic of it, and have no necessary relation, in their various alterations, to those of the vesicular type of breathing sound. That is, the student may remember that such an acoustic law, for instance, as the lower the pitch the louder the sound, *ceteris paribus,* holds for either the bronchial or the vesicular breathing sounds, respectively; but it may not do so interchangeably under either normal or pathological conditions, since the qualities are entirely different, and these are fundamental. This statement is made here to anticipate the confusion of mind which I have witnessed in many students at the seeming contradiction of high-pitched, loud bronchial breath sound and comparatively low-pitched vesicular murmur of soft intensity: these are their respective characteristics, regardless of any relation of the variable attributes of one to the other, while such variations (normal or pathologic) of either are, of course, subject to the laws of sound already indicated.

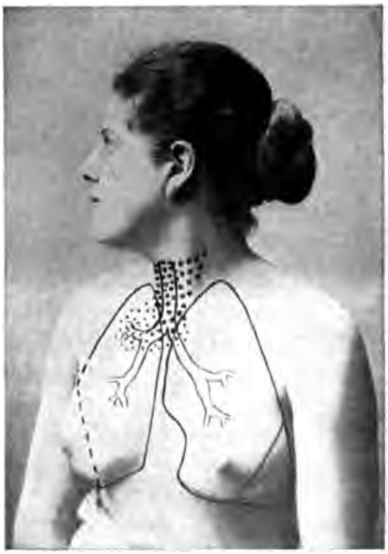


Fig. 36.—Dotted Area shows where Bronchial (larger dots) and Broncho-Vesicular (smaller dots) Breathing Normally Exist. Note higher origin of bronchus to right upper lobe compared with that to left upper lobe. Shows also line of demarcation between upper and lower lobes of left lung anteriorly. (Butler.)

Bronchial breathing is also heard in the interscapular space, near the root of the lungs. It is produced by the impartation of motion to the tracheobronchial column of air by the eddylike currents set up during inspiration at the rima glottidis. Therefore, it is here, over the larynx, that the breath sounds have the loudest, harshest quality. Care should be taken not to be deceived by the pharyngeal sound sometimes very audible while the person examined breathes with the mouth closed, nor by the lip murmur sometimes caused, as when the mouth is open.

Normal variations are met with. Thus, the sound is often harsher when the mouth is closed. The deeper the breathing the louder the sound. It is more distinct over the right than over the left bronchus, near the tracheal junction. The superior boundary of its audibility is the seventh cervical vertebra. In persons with thick, heavy chest walls, the intensity of the bronchial sound may be decidedly diminished, but the tubal, piping quality, and high-pitched, relatively prolonged expiration is nevertheless characteristically audible.

(2) **Normal bronchovesicular breath sound** is an imperfect variety of bronchial breathing, having some of the attributes of the vesicular or true lung sound without having lost those of the bronchial; it is an intermediate, indeterminate, or mixed respiratory sound, on the borderline between bronchial and vesicular respiration. It is heard especially over the right apex, including the right infraclavicular region; also in passing with the stethoscope from the trachea and one of the primary bronchi over the sternum, downward and outward for a very short distance, variable in different individuals, barely more than 1 to 2 in., until the vesicular type of lung parenchyma is reached unmodified by the predominance and largeness of caliber of adjacent bronchial tubes.

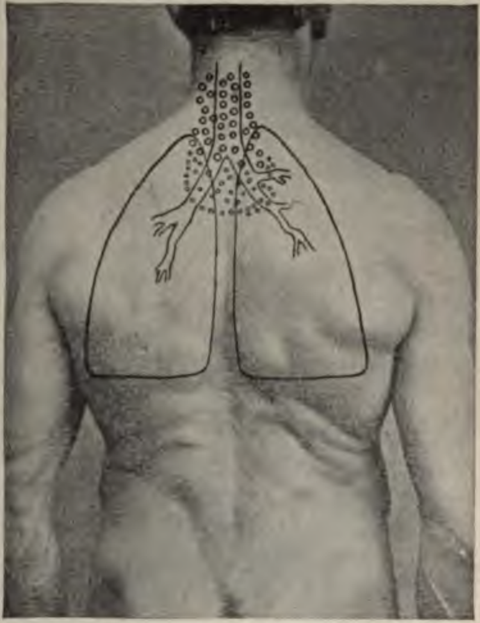


FIG. 37.—SHOWING THE SITE OF NORMAL BRONCHIAL (LARGE DOTS) AND BRONCHO-VESICULAR (SMALL DOTS) RESPIRATION POSTERIORLY. (Butler.)

The bronchial element mixed with the vesicular which the bronchus on the right side imparts in the infraclavicular region is a feature of considerable practical importance which, when taken in connection with the other normal physical signs in this region, may easily be mistaken for the physical evidences of the early stage of tuberculosis, as compared with the left side. The expiration is a little harsher, higher in pitch, and longer than that of the vesicular breathing, but a little less pronounced in these characteristics than in the bronchial breathing. Inspiration is also slightly feebler, relatively longer, and lower pitched than the bronchial sound.

(3) **Vesicular Breathing.**—This is typical normal lung sound, heard wherever the larger bronchi are not within auscultatory distance; that is, it is audible over every portion of the chest wall corresponding to the surface of the lungs, with the average exception of the right infraclavicular space inside the midclavicular line and the interscapular region to the third or fourth dorsal vertebra. It is most distinctively heard in the infrascapular region, in good breathers in the axillary regions, and in the left infraclavicular region not too near the left bronchus. Its characteristic QUALITY is breezy, rustling, like the sighing sound produced at a distance by leafy tree branches in a grove swayed by a gentle wind, whereas the bronchial sound may be likened more to the piping swish of a wintry blast through the bare boughs of a forested plot. The vesicular sound may be imitated most nearly by placing the lips, almost closed, in the position to say " f " or " v," and then gently drawing a current of air inward, as for the " f," to reproduce the inspiratory portion of the sound, while the short expiratory sound is simulated better by the " v " characteristic in letting the breath escape.

The INTENSITY of the vesicular sound varies in different individuals, being generally softer and feebler to a marked degree as compared to the tracheal or bronchial breath sound, and in some instances hardly audible at all. The inspiratory part of the sound is louder than the expiratory. It does not commence in, but ascends to its maximum intensity, which is not quite maintained, however, up to the end of the act, or the beginning of expiration; on the other hand, the expiratory sound is loudest at its commencement, and dies away so rapidly that it ceases to be heard throughout nearly the latter three-fourths of the expiratory relaxation of the alveoli and recoil of the chest wall.

Compared with the PITCH of laryngeal or bronchial breathing, that of the vesicular murmur is low. The expiratory sound is usually lower in pitch than the inspiratory, although in some cases, as pointed out by a few authors, it may be slightly higher; I have found this to be so principally in children.

The relative DURATION of inspiration and expiration is characteristic of the vesicular breath sound; that is, the former is three or four times longer than the latter. Right here a careful discrimination must be made to avoid a seeming contradiction between the physiologist and the physical diagnostician, and consequent confusion in the student's mind. For, as a physiologic function or act, the ratio of inspiration to expiration is about as 5 to 6, in relative lengths; whereas, as a clinical fact, their relative durations of audibility are as 3 or 4 to 1. The inspiratory sound is heard with sufficient length to indicate its breezy quality, while the expiratory sound is heard, if at all, as a mere soft puff or whiff of air. Thus it is that absence of the expiratory murmur is practically never pathologic, a relative lengthening, on the other hand, accompanying nearly all of the morbid conditions of the lung.

An important feature of normal vesicular respiration is the very short pause between the inspiratory and expiratory portions of the act, the sounds following each other so closely that they are virtually continuous. The nearer one approaches regions of the production of bronchial sound, whether normal or abnormal, the more distinct on auscultation becomes the pause between inspiration and expiration.

The RHYTHM or rate of recurrence of the sounds of inspiration and expiration is characterized normally by regular intervals.

ORIGIN.—Opinions vary as to the precise cause of the sound of vesicular or cell breathing. None of the theories proposed have received, as yet, demonstration by positive proofs.

According to Laennec and others, the sound is produced by the entrance of the air into, and its expulsion from, the air-cells—by the friction of the air in its passage in and out of the bronchioles and alveoli, against their walls. The more probable hypothesis of Baas, Penzoldt, and perhaps the majority of writers and experimenters, attributes the vesicular murmur to the modified laryngotracheal sound transmitted downward with reduced force and increased diffusion through the branching and diminishing capacity of the bronchial tubes, and softened by the distant, dissipating effect of the vibrating and absorbing spongy vesicular lung tissue. That the vesicular breath sound is really the bronchotracheal sound modified by its conduction and prolongation into the bronchial ramifications and alveoli may be shown in the fact that when the latter are deprived of air by the disease products in consolidated lung, as in pneumonitis, the harsh tubular breathing is transmitted readily to the chest wall, and thus to the ear, the muffling effect of the air-cells being in abeyance, and the intensifying effect of the solidified lung being superadded. At the same time, it cannot be truly denied that, if only to a minor degree, the vibrations at the terminal portions of the bronchi, at the instant of vesicular dilation, may be contributory factors in the production of this respiratory murmur.

As the larger bronchial tubes branch off into the smaller, and the latter into the bronchioles, not only is the harsh, blowing character of the bronchial sound softened and damped by the consequent diffusion and interfering reflection of weakened and superadded aerial vibrations, but also by the gradual structural changes in the walls of the tubes. That is, as the homogeneity of structure of the larger bronchial tubes, with their cartilaginous and muscular walls, becomes lost in the transition to the elastic membranous bronchioles and alveoli, the pronounced tubular quality and intensity become diminished and confused, owing to the loss of conductivity of the tissue vibrations of the latter as compared with the former.

The reason why the expirat[illegible] d is not so long nor so loud as the inspiratory is probably be[illegible] the latter instance the vibrating current of air is directed tow[illegible] auscultating ear, and in the former, from it. This is augmented [illegible] the fact that the inspiratory movement is normally more energe[illegible]han the expiratory; it is the combination of muscular and atmosph[illegible]c force in the one case against the simple recoil and relaxation of the a[illegible]r vesicles. Again, the tension of the alveoli during inspiration rend[illegible]s the breath sound louder at this time because of the greater conductivity of their tissue.

NORMAL VARIATIONS.—Quite compatible with a normal state of the lungs, there are certain peculiarities and variations of vesicular breathing which need to be recognized. The first of these is:

Age.—In children under about the twelfth year, the vesicular murmur is exaggerated, harsher, and louder than in adults of middle age. This increased intensity is characteristic of the expiration as well as of the inspiration. As will be pointed out later, lung that is doing extra or compensatory work, because of another portion of lung that is disabled by disease, produces an exaggeration of the vesicular breath sound which, resembling the normal quality and intensity of the breathing of childhood, is often spoken of as ***puerile respiration.*** That this peculiarity is due to the relatively smaller and more elastic vesicles, with consequent larger relative proportion of bronchial tissue, and more resistance to the inspiratory effort calling for more forcible action, both combining to roughen the murmur, is quite obvious. Undoubtedly, an additional factor, passive, is the very trifling opposition to the transmission of sound which the thin, elastic chest walls of young children offer.

In old age, on the other hand, the *intensity* of the vesicular respiration is diminished, while, owing to the weakened and inelastic condition of the lung, the inspiration is slightly shortened and the expiration a little prolonged, the alveoli recoiling tardily and slowly. *Senile respiration*, as observed in healthy old people, is to be differentiated, however, from the *morbidly weakened* respiration in the greater length of the expiratory sound of the former.

Sex.—Generally, the total respiratory murmur in the female is louder than in the male, especially in the upper, anterior regions of the chest.

Region and Side of the Chest.—In most healthy individuals the sounds are more distinct and intense on the left side, and particularly in the infraclavicular region. They are, of course, more audible where the chest walls are thinnest; hence, they are clearer anteriorly than posteriorly, over the axillary and infrascapular regions than the mammary and scapular. It is of such great practical importance, that to repeat here what was indicated before concerning the bronchovesicular character of the breath sounds near the junction of the second rib with the sternum on the right side, due to the closely subjacent bronchus, is not at all irrelevant. The harsher blowing and more prominent expiratory element in the interscapular region must also be reiterated in this connection. The vesicular murmur is relatively weak, too, over the apices, with their small volumes of air capacity. Not only increased thickness of the soft parts, as in obesity, but also massiveness and increased convexity of the ribs, interfere decidedly with the ready transmissibility of the vesicular sounds.

Rapidity, Fulness, and Continuity of Respirations.—The intensity of the respiratory sounds increases directly with the frequency of the breathings. Also, the duration of both inspiratory and expiratory sounds is increased with full and deep respirations, as after exercise or excitement. Marked intensity of the respiratory murmur may be a matter of temperament, idiosyncrasy, or of some neurotic disposition, as in hysteria.

A *jerky, interrupted*, or *wavy inspiration*, though frequently associated with incipient tuberculosis, may be heard as a special peculiarity consistent with perfect pulmonary health. It occurs in certain persons who breathe slowly and irregularly, and may be caused to disappear after a quick, full inspiration. Physiologic jerky respiration is heard nearly uniformly over the lungs; that due to tuberculosis is limited to a small area, usually one or other apex. It may be found, also, in hysterical persons, and in whining or sobbing children. It is recognized by the inspiratory murmur occurring in a series of short puffs, with scarcely more than two or three intermissions. The character of interrupted breathing may be simulated by pursing the lips and drawing the air through them in several quick, sudden draughts.

Systolic vesicular breathing refers to a rhythmic exaggeration of the respiratory murmur, principally during inspiration, also jerky or puffy in quality, coinciding with the cardiac contraction, and heard at the border of the lung near the heart. As the lung expands, with the alveoli in closer contact with the heart, the systolic movements of the latter cause an unequal entrance of air into the vesicles—a rhythmical rush corresponding with the systole, increasing in intensity up to the end of inspiration, and rapidly subsiding at the beginning of expiration.

I have known this phenomenon in not a few cases to be mistaken for a valvular heart murmur, either by students or by examiners doing rapid, desultory dispensary work.

Having become familiar with the normal sounds of respiration, and with their normal variations and peculiarities, one is ready to apprehend and analyze the pathologic sounds.

SOUNDS OF ABNORMAL RESPIRATORY CONDITIONS

Auscultation determines, first of all, the pathologic modifications of the breath sounds, and essentially of their attributes of *intensity*, *rhythm*, and *quality*. The examination is practically confined to those regions of the chest which cover the lungs; that is, where vesicular respiration or, as over the right apex or interscapular region, bronchovesicular breathing is normally audible. So long as we hear the vesicular respiratory murmur over the chest, we are assured that the subjacent air vesicles are permeable, as well as that the supplying bronchial tubes must likewise be free.

The student's first aim is to satisfy himself that he has eliminated the confusing effect of the heart sounds; that he is listening exclusively and definitely to the respiratory murmur; and then, that he can clearly distinguish the sounds corresponding to the two acts of respiration. The latter may be facilitated by placing the hand upon the thorax or epigastrium.

Having fixed the attention on the inspiratory and expiratory sounds, it is of prime importance to analyze carefully their (*a*) *loudness* or *intensity*; (*b*) their *relative lengths*, and the presence and duration or absence of a *pause* between them; (*c*) the *character* or *quality* of both sounds; (*d*) the *pitch*, especially of the expiratory sound.

(A) *Pathologic Modifications of the Vesicular Breathing*

Alterations of Intensity. (1) INTENSITY INCREASED: *Exaggerated, Compensatory, Vicarious, or Puerile Breathing.*—Exaggerated respiration differs from the normal in being louder, and at the same time longer (for it is quite common to have two or more attributes suffer alteration simultaneously). The increased intensity affects both the inspiratory and expiratory sounds, but is especially noticeable of the expiratory, as normally this is scarcely heard at all. The inspiratory sound is, to be sure, predominantly much louder, and the increased audibility of expiratory sound is partly because of its longer duration. On account of its superficial resemblance to pathologic bronchovesicular breathing, the unskilled examiner may erroneously attribute morbid changes to a portion of lung that is simply overacting. An important element in the differentiation here lies in the fact that while the respiratory murmur is lengthened, the normal ratio is maintained; that is, the duration of inspiration to expiration is as 6 to 2 (normal, 3 to 1). The pitch remains unaltered. The vesicular quality, though harsher—like the *puerile* respiration of children—nevertheless lacks the bronchial modification indicative of closure of the alveoli; the softer vesicular quality is still preserved.

The immediate *cause* of exaggerated or intensified breath sound is increased functional activity of the whole or a part of a lung, compensatory or supplementary to restricted or abolished action due to disease of the opposite, or another part of the same lung. Hence, the discovery of exaggerated breathing is the indirect evidence of some morbid pulmonary condition elsewhere.

It occurs over healthy lung tissue adjoining parts where the smaller bronchial tubes are partially obstructed by the tumefaction of an acute catarrhal inflammation of the mucous membrane, aggravated by an accumulation of sticky mucus. The appearance of a heightened intensity or sharpness of the inspiratory sound over a lung apex may be the first auscultatory evidence of a beginning tuberculous bronchitis—hardly more than a tiny tubercle projecting into the lumen of a bronchiole so as to cause compensatory increase of force of breathing in the surrounding vesicles. *Unilateral* exaggeration of the breath sounds is typically heard in cases of severe pneumonitis affecting the opposite lung, or of compression of the other lung by a large pleuritic effusion. *Local* intensification of the vesicular respiratory murmur may be present over the upper lobe of a lung that is relaxed in its lower portion by being floated up by a small or moderate pleural effusion, or over either the upper or lower lobe when the other is the seat of a pneumonic consolidation. Again, in tuberculous consolidation of one apex, exaggerated vesicular breathing of the *opposite* apex is commonly noted.

Finally, it should be mentioned that exaggerated breathing over the upper portion of both lungs is a frequent accompaniment of tightly laced women.

(2) INTENSITY DIMINISHED: *Weak, Shallow, or Senile Breathing.*—This is a soft and distant degree of loudness, a simple weakness of the intensity of the vesicular respiration. At the same time, the duration of both inspiratory and expiratory sounds is proportionately shortened. The inspiratory sound is distinctly weakened, and the expiratory sound is practically inaudible.

The numerous *causes* of feeble respiration may be considered under three heads, as follows: (1) Those which interfere with the perfect transmission of the breath sounds; (2) those which prevent full expansion of the lungs, (3) or which diminish the elasticity of the vesicles.

Of the first condition, we have edematous thickening of the chest walls, obesity, or some solid tumor; principally, however, it is due to some thickened or adherent pleura, or to a moderate pleural liquid effusion; in the case of the latter, the faintness of the respiratory murmur is usually on one side of the chest, while on the unaffected side relative exaggeration of breathing may be heard. Pneumothorax (air in the pleural sac) of moderate degree, or circumscribed by adhesions, may also intervene between the lung and chest wall, so as to weaken the breath sounds without modifying the vesicular quality.

Secondly, conditions which interfere with expansion of the lung (besides those which are referred to in the preceding paragraph, and which so act indirectly) are mainly the following: (*a*) Obstructions of the larynx, trachea, or a bronchus, as from foreign body within, pressure of a tumor or aneurism from without, inflammatory or membranous swelling or edema of the larynx or trachea, accumulations of mucus and pus, and catarrhal thickening of the bronchial mucosa in bronchitis and bronchiolitis; also from spasm of the larynx, and of the bronchioles in asthma, and from partial filling of the alveoli in pulmonary edema. (*b*) Deficient respiratory action, as from rigidity of the thorax, paralysis of the diaphragm or thoracic muscles, painful conditions such as pleuritis, pleurodynia, or intercostal neuralgia, and general peritonitis; again, from mechanical restraint due to abdominal enlargements, as ascites, tympanites, and large tumors.

In the third place, diminished elasticity of the vesicular tissue from the weakness of permanent overdistention is characteristic of emphysema, in which feeble respiratory murmur is a constant physical sign. In the first, or congestive, stage of acute lobar pneumonitis, also, shallow breathing may be noted.

In the last-named affection the weak breathing is heard on both sides of the chest. *Unilateral* diminution of loudness is a feature of partial occlusion of one or other bronchus, pleural effusion, and the early stage of pneumonitis, although in cases of the latter, as with tuberculous consolidation, the diminution may be only *local*, depending upon the extent of involvement. Where the movements of the thorax as a whole are restricted, as from the conditions other than emphysema mentioned, *bilateral* enfeeblement of respiratory sounds may be observed. In connection with the painful conditions, as pleuritis of the dry, plastic variety, and pleurodynia, the deficiency of breathing may vary and thus assume an *intermittent* form.

(3) Absent or Suppressed Breathing.—This occurs over large pleuritic exudations, as of pus, serofibrin, and blood; over diffuse, closed pneumothorax, with complete occlusion of either bronchus, and over great thickening of the pleura, or of the lung in fibroid phthisis. The breath sounds may be abolished also from any aggravation of the conditions which produce mere weakening of the vesicular respiration. Obviously, in the complete infiltration of the air-cells which follows the congestive stage of pneumonitis, and in extensive tubercular deposit, the abolition of the vesicular murmur is due to its replacement by a changed type—bronchial or bronchovesicular—of breathing.

Alterations of Rhythm.—These have to do with disturbances of the regularity, continuity, and relative lengths of the inspiratory and expiratory sounds, and of the pause between them.

(1) Interrupted, Jerky, or "Cogwheel" Respiration.—The occurrence of this kind of breathing as a normal variation has been referred to before. Therefore, too much importance must not be given to it as to its diagnostic value in pathologic conditions, and yet the discovery of it over a lung apex should awaken suspicion as to the presence of a tuberculous bronchiolitis or localized pleuritis.

Usually the inspiratory sound, instead of being even and continuous, is broken into a series of puffs separated by irregular, short intervals, hence the term "cogwheel" respiration. It is at the same time rough or harsh in incipient tuberculosis. Aside from its occurrence in nervous, chilly, or fatigued persons (from irregular muscular action), it should not be confused with the cardiorespiratory whiffing sound associated with excited cardiac movements. The latter is more likely to be localized adjacent to and a little beyond the left border of the heart; in cases of the former, the interrupted breathing is generalized. I have found the cardiopulmonary variety of puffy respiration to be a trifle sharper, and prompter in its onset than that due to apical bronchiolitis.

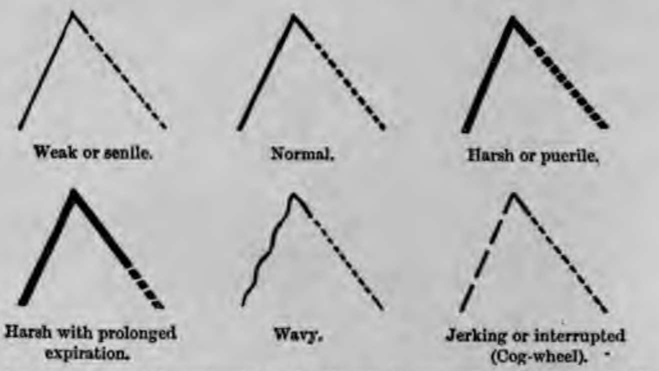

Weak or senile.
Normal.
Harsh or puerile.
Harsh with prolonged expiration.
Wavy.
Jerking or interrupted (Cog-wheel).

Fig. 38.—Diagrammatic Representation of the Varieties of Vesicular Breathing. The dotted part of down line represents the inaudible part of expiration. After Wyllie. (Gibson and Russell.)

Pathologic *wavy* or *jerky* breathing is most commonly the result of tubercles protruding from the wall of an infiltrated bronchiole, or of the presence of tenacious mucus, both interfering either with the continuous passage of the air current directly, or causing adjacent lobules to expand at different times. Associated with other confirmatory physical signs, interrupted respiration thus becomes an indication of early tuberculosis, localized over the affected apex. The same effect upon the breath sound may be produced there by a circumscribed area of adhesive pleuritis of tuberculous origin.

As a case of tuberculosis progresses, the type of breathing changes to the bronchovesicular, with prolonged expiration, when the sound of the latter may also be wavy, or interrupted and jerky.

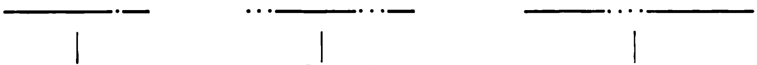
In the first stage of acute plastic pleuritis, in pleurodynia and intercostal neuralgia, cogwheel respiratory rhythm may be heard on the side affected.

(2) Lengthened Interval between Inspiration and Expiration.—Instead of the inspiratory and expiratory sounds succeeding each other with the seeming closeness of continuity, pathologic prolongation of the momentary pause between them may be discovered; hence the term "*divided respiration*" (Walshe). When this occurs,

it is either because the *inspiratory* sound is *shortened*, unfinished, or because the *expiratory* sound is *delayed*, deferred.

Lengthened pause from delay in the oncoming of the expiratory sound is characteristic of emphysema, with its impaired elasticity of the vesicles, the first part of the expiratory act being unaccompanied by sound.

Shortened inspiratory sound is observed in cases of marked consolidation of the lung, as from pneumonitis or tuberculosis, the inspiratory sound ceasing before the chest expansion, the breathing being harsh or bronchial. More frequently, however, we meet with a deferred inspiration in emphysema, also, the inspiratory movement of the chest commencing before the sound becomes audible.



Normal. Shortened inspiration. Delayed expiration.

FIG. 39.—DIAGRAMMATIC REPRESENTATION OF PROLONGED PAUSE.

(3) PROLONGED EXPIRATION.—As the normal expiratory sound is so short that it is scarcely more than audible, any *relative* increase in its duration must necessarily be abnormal. The qualifying term relative is used with emphasis, indicative of the disproportionate lengthening of the expiratory sound, so that the normal ratio of inspiratory to expiratory duration, 3 to 1, becomes 3 : 2, as in broncho-vesicular breathing, or 3 : 3, as in bronchial breathing. Even the prolonged expiration of exaggerated respiration, where the normal ratio of duration is maintained by a corresponding lengthening of inspiration, hints at the presence of disease in some other portion of lung.

But the significance of prolonged expiratory sound is not specific or independent; on the other hand, it depends upon its amount of relative increase, and associated alterations of pitch especially, and of quality. Thus, when harsh and tubular in quality, high in pitch, and as long, or even a little longer, than the inspiratory sound, having a ratio of 3 to 4, it means that the vesicles of the area involved are filled with a tuberculous or inflammatory deposit of nearly or quite lobar size, so that air is free to pass in and out of the bronchial tubes only; hence the sound has the characteristics of bronchial breathing. Again, with a still longer relative increase in the expiration, as 1 : 3, but with feeble vesicular quality of low pitch, as is heard over both lungs in emphysema, the cause of the prolongation resides in the loss of elasticity due to a permanent and, as it were, paretic condition of the dilated alveolar walls. Moreover, in emphysema we have another cause for the prolonged expiratory sound in the obstruction to the free egress of air on account of the diffuse bronchial catarrh nearly always associated. Lastly, emphysema is the only condition in which the prolonged expiration is *low* in pitch; all other causes of this change produce at the same time elevated pitch, with the occasional exception of a deeply seated, lax-walled cavity in the lung, the breath sounds over which may have a low-pitched, hollow quality.

By way of antithesis and association, the two principal physical changes in the lungs may be placed thus:

Consolidation	*Emphysema*
Vocal fremitus; increased.	Diminished.
Percussion; dulness, high-pitched.	Hyperresonance, low-pitched.
Auscultation; expiratory sound prolonged, high-pitched, harsh, bronchial.	Also prolonged expiration, but low-pitched, feeble, vesicular.

When due to asthma, the prolonged expiratory sound may be obscured by the loud, dry, wheezing or whistling sounds or râles, because of spasm and mucous clogging of the bronchioles.

The occurrence of slightly lengthened and higher-pitched expiratory murmur over an apex, with or without adventitious râles, is one of the positive physical signs of beginning tubercular infiltration there—bronchovesicular breathing.

A harsh, prolonged expiratory breath sound may also be the result of a severe general bronchitis, as in children who have had measles or whooping-cough preceding it, or in very old people subject to "winter cough," and having weak hearts. The extensive bronchial catarrh is distinctly indicated by the evident obstruction to the exit of air currents that apparently have difficulty in entering the vesicles.

Alterations of Pitch.—The pitch of normal vesicular respiration being low, all elevations of this attribute point to bronchovesicular or bronchial modifications, according to the extent of tissue consolidated. The change is most significant in relation to the expiration, which is always simultaneously prolonged with the abnormally high pitch. The low-pitched cavernous breathing sometimes met with also has a quality of sound that is not easily confused with the vesicular type.

Alterations of Quality.—Wherever the normal vesicular murmur is replaced by another quality or type of breath sound, the recognition and discrimination is made by an analysis of the combination of the other changed elements. The name given to the pathologic substitute for the normal vesicular respiration is of secondary importance, and may be only a vague and convenient intimation of the actual determining factors. In short, we make the diagnosis of morbid physical changes in the lung by a careful, penetrating comparison of the changed attributes rather than by an endeavor to embrace and describe them under a name, either to oneself or to others. One should rigidly and critically define the expressions used below in terms of the altered conditions of rhythm, intensity, and pitch just given in the preceding paragraphs.

(1) BRONCHOVESICULAR, HARSH, OR RUDE RESPIRATION; INDETERMINATE BREATHING.—In this variety of breathing, the soft, breezy quality of the normal vesicular sound is lost and exchanged for a harsher, sharper, more blowing sound, more marked in expiration than in inspiration. And yet the sounds of both are not as rough and tubular as in bronchial breathing. Naturally, there may be many degrees of variation between true vesicular and bronchial respiration; therefore the terms "indeterminate" or "transition," sometimes applied to this type, especially by European clinicians. It is detected over the small areas of condensation of lung, not too deeply situated, where it is caused by the generally sharp and harsh respiration, and particularly by the relative prolongation and elevation of pitch of the expiratory sound.

The physics of the pathologic source of the bronchovesicular breathing is easily understood: A certain number of vesicles infiltrated with firm exudate form a lobular area of solidification of better conductivity for the included bronchioles, but not large enough to prevent the surrounding open vesicles from subduing the tubular, bronchial harshness of more extensive areas of consolidation. Bronchovesicular breathing may be heard also over large consolidations with some thickness of unaffected lung intervening.

If bronchovesicular respiration be persistently audible at one apex, especially the left, while at the other ordinary vesicular respiration remains, incipient tuberculous infiltration may be inferred, the indeterminate breathing being produced partly by mucous obstruction of some of the bronchioles or smaller bronchi, as well as the restricted expansibility of the alveoli. Right here the physiologic difference between the two sides must not be forgotten—that at the right apex, he s clavicular and supraspinous regions, bronchovesicular breath sound is normal. This is particularly noticeable during quiet breathing in robust and muscular individuals, the air-current enter- g the vesicles so weakly and the thick thoracic tissues transmitting its characteristics so slightly that the bronchial element is a little dominant, and the vesicular character becomes fully brought out y with deep respirations.

Bronchovesicular breathing may be indicative of patches of col- nsed lung (*atelectasis*) in the acute capillary bronchitis of children. ilarly, it is found over small areas of bronchopneumonitis, espe- y in the infrascapular regions of both sides, in the very young the aged. In the atypical pneumonites or pulmonary congestions plicating influenza ("grip"), the occurrence of indeterminate athing is almost constant. Cases of frank, typical lobar pneu- onitis in adults, also, in the first or congestive stage, give rise to onchovesicular respiration.

The *differential* physical diagnosis between simple exaggerated reathing and the bronchovesicular variety is of extreme practical rtance, and may be a matter of difficulty, doubt, and error to ician of limited experience as well as to the more excusable student tyro. The superficial resemblance which the two types of respiratory sound have to each other, and which may confuse the inexperienced, lies in the increased loudness and increased length of the inspiratory and expiratory sounds. But the inspiratory element of bronchovesicular breathing has a quality of sharpness, of dry harshness, suggestive at once of a small area of fine-calibered tubes instead of the softer, more breezy loudness suggestive of a more voluminous area of spongy, elastic air-cells. More distinctive, however, are the differences concerning the expiratory sound. While this is prolonged in both the exaggerated and bronchovesicular breathings, in the latter the normal ratio is broken; that is, as pointed out before, the expiratory sound is abnormal in its relative or proportionate length to the inspiratory sound. This is by all means the principal differential sign. Next in value is the harsher, higher-pitched expiratory sound.

To mistake bronchovesicular breathing for exaggerated or puerile breathing is to assume the absence of a diseased portion of lung where there actually is disease; to reverse the error is to assume or infer the presence of a small area of consolidation which does not exist in that region, but either adjacent to it or in the opposite lung; perforce, to fail to detect either type of respiration is to fail to discover either *directly* or indirectly any limited area of solidification.

(2) **Bronchial, Tubular Respiration.**—Here there is a complete absence of the vesicular quality, and wherever substituted for the latter is pathologic. It may be recognized readily by comparing its attributes with the ever-present normal bronchial breathing heard over the trachea. Indeed, the bronchial breathing, caused as it is by *large consolidations*, is not essentially pathologic, but is merely the sound of the deeply seated, normal bronchial breathing heard through the pathologically infiltrated vesicles, which now form an area of good conducting, solid tissue. It is not a superadded type of respiration; it is simply an abnormally transmitted normal type.

Distinct bronchial breathing resembles more nearly the blowing sounds heard at the bifurcation of the trachea. It is rather harsh, loud, and tubular, but not quite so much so as the bruit heard over the larynx. Both inspiration and expiration are *higher in pitch* than of vesicular breathing, the expiration frequently even higher than the inspiration, depending, on the whole, upon the force of the respiratory act and the caliber of the bronchial tubes. The inspiratory sound is a little shorter in duration than the inspiratory act, is rhythmically separated by a short interval from the expiratory sound, which is longer than that of vesicular breathing, and fully as long as the inspiratory sound. The *intensity* is marked, but variable; it may have the harsh, dry, loud, whiffing character simulated by blowing to and fro through a metallic tube, as over a densely consolidated portion of lung near the ear of the auscultator, and conducting the sound from a few large tubes, perhaps; or it may be less loud, more diffuse, over moderately consolidated, less superficially situated tissue, and transmitted from a number of smaller tubes. At the same time it should be noted that, while in the former instance the *pitch* may be lower than in the latter, great rapidity of respirations may cause a higher pitch than over the moderately solidified lung with softer bronchial blow. As a rule, however, the higher the pitch of the bronchial breathing the more intense the degree of consolidation, exemplified especially in the most acute cases of lobar pneumonitis, while the medium pitch is more characteristic of large tuberculous consolidations. Of course, the larger the part hepatized (firmly infiltrated, liver-like), the louder the bronchial sound, because the greater the volume of bronchial air-space within.

In order to appreciate the diagnostic significance of pathologic bronchial respiration, therefore, it is necessary to realize clearly the following fundamental considerations: Any breathing sound heard at all means open bronchial tubes; a vesicular breathing sound means open alveoli; a bronchial sound indicates that there is closed vesicular lung tissue between the bronchial tubes and the ear of the auscultator, the tidal movement of air being prevented either by *exudation within the vesicles, or by compression from without.*

(*a*) As intimated before, lobar pneumonitis is the typical cause of bronchial breathing, the condensation of lung tissue involving an extent sufficiently large so as to include the medium-sized or large bronchial tubes, bronchovesicular respiration resulting when only the smallest tubes are surrounded by relatively smaller areas of thickening.

In some cases of pneu ver, owing to occlusion of the
large supplying bronchus of the smaller tubes (inflam-
matory exudate; mucus, eath sound is heard; but after
a loose cough the tubes ious, and bronchial breathing
may then be transmitted. onia usually attacks the lower
lobes, bronchial respiration hea e *base* of the chest posteriorly,
and up to a little above the angle he scapula, in an acute illness,
signifies *pneumonia* most frequ At the *apex*, tuberculosis is
the common cause of bronchial b ng in the moderately advanced
cases, although apical pneumonit y rarely be present.

Among other and less common ditions of solidification of lung tissue giving rise to bronchial breathing are *chronic fibroid pneumonia* (phthisical or syphilitic), *hemorrhagic infarcts*, and, less perfectly and under favorable circumstances, *central pneumonitis* (followed by more marked bronchial breathing usually within a week, as the consolidation extends to the surface), carcinomatous and other *new growths*, and *pulmonary abscess* and *gangrene.*

(*b*) *Compression of the lungs* by a large pleuritic effusion, so that the vesicles are void of air while the bronchi remain patulous, gives rise to bronchial breathing, also. It is in the majority of cases distant and feeble, except near the spine, where the lung is collapsed; it is also more decidedly audible at the upper layer of the fluid. Care must be taken that distant bronchial respiration be not mistaken for bronchovesicular. Directly over the exudation the respiratory sounds are inaudible. The presence of a pleuritic effusion complicating a pneumonitis will have the effect of similarly enfeebling the underlying bronchial breathing produced by the consolidation, the change of consistence of the media having a damping effect.

Pneumothorax not infrequently causes such a compression of the lung as to produce bronchial breathing heard best between the shoulders. Partial collapse of lung may also make pronounced the bronchial element due to large *pericardial effusions*, large *aneurisms*, great *enlargement of the heart,* and the pressure of *malignant growths* within the pleural sac.

NOTE.—It may not be amiss to repeat here the caution not to infer the presence of pathologic bronchial breathing owing to the ready transmission of nasopharyngeal respiration. This fallacy may be obviated by requesting the patient to breathe deeply, but continuously and unconstrainedly. It is extremely unsatisfactory to have patients, when told to breathe fully, take a deep inspiration, then hold it for a few seconds, with rigid chest, before slowly, and often very much too slowly, allowing expiration to take place. The actual presence of physical conditions productive of bronchial breathing may be obscured in this way, the quality of the expiratory sound unduly softened, and the true relative lengths of the inspiratory and expiratory sounds artificially altered. Instructing the patient to increase the rapidity of the respirations, with the mouth open, will usually correct the mode.

Cavernous respiration, though heard over cavities of the lung, and thus significant of distinctive conditions, nevertheless so closely resembles the physical characteristics of bronchial breathing that it may properly be considered under this head.

Laennec, who introduced the term, described cavernous breathing as giving to the auscultating ear the impression of a bronchial air-current passing promptly into a large, hollow space. Perhaps the post-mortem conditions discovered were largely influential in framing this description. The distinguishing characteristics must be recognized in the modified attributes of bronchial breathing. These are noticed to develop in observing the course of a case of progressive tuberculosis of the lung, in passing from the consolidation stage to that of cavity formation.

The previously harsh, piping *quality* of the bronchial breathing becomes softer and hollow; the *pitch* is low, especially of the expiratory sound—a distinguishing feature, according to the elder Austin Flint, in that the expiratory sound of true bronchial respiration is usually higher in pitch than the inspiratory sound. As to *rhythm*, the expiratory has nearly the same length as the inspiratory sound. Other elements of contrast which aid in physical diagnosis are as follows: Cavernous breathing seems remote from the examiner's ear, less intense, while bronchial breathing is more distinct and superficial; the former is heard over a more limited, circumscribed area, the latter over a diffuse, irregularly outlined area; lastly, the production of the cavernous breath sounds is slower, retarded, that of the bronchial more prompt and decided.

These points of differentiation are observable mostly where the simple condition of a globular cavity with flaccid walls exists, large enough, superficial enough, and in communication with a bronchial tube. In general, and other things being equal, the *intensity* of the cavernous respiratory sound depends upon the situation and size of the pulmonary excavation, the caliber of the communicating bronchus, the freedom from occluding exudate, and the energy of the respiratory act.

In the event of the cavity being surrounded by solidified lung, as is quite frequently the case, before the focus of caseation and excavation has extended far, the bronchial and cavernous characteristics are so intermingled (*bronchocavernous breathing*) that it becomes difficult to determine precisely the actual physical changes. Again, should a sufficient thickness of healthy vesicular tissue lie between the cavern and the chest wall, the bronchial quality of the respiration in the cavity loses its distinctive character in the feeble conducting power of the alveolæ (*vesiculocavernous breathing*). And finally, cavernous breathing may be weakened by temporary narrowing or occlusion of the bronchus leading to the cavity, or by a partial filling of the latter with muco-pus, or lost by complete plugging of the bronchus or filling of the cavity; in either instance, the typical breathing may be re-established after cough and expectoration.

Besides the commonest cause of cavernous respiration, namely, tuberculosis of advanced stage (usually at the apices), it may be heard over cavities the result of *abscess, gangrene, cancer,* or due to a *dilated bronchus.*

Although of rare occurrence, it sometimes happens that cavernous respiration is audible in the absence of pulmonary cavity. This has been observed in certain cases of consolidation surrounding a large bronchus in pneumonitis, and of pulmonary collapse from pleuritic effusion, the subsequent course disproving excavation of the pulmonary parenchyma by the disappearance of the signs with recovery.

(3) AMPHORIC RESPIRATION.—This has a metallic, echoing, hollow, or blowing quality, and therefore is more easily distinguished than the cavernous respiration. As the appellation indicates (*amphora, a jar*), it may be imitated by blowing gently across the mouth of an empty bottle or jar. Amphoric breathing is analogous to the amphoric resonance obtained on percussion over large, empty, dense-walled pulmonary cavities.

In some cases amphoric breathing is quite loud; in others it may be audible only on deep or forced breathing. The pitch varies according to the volume of contained air, and the tension of the cavity walls; the larger the lower the pitch, and the tenser the higher the pitch, although the walls are characteristically distended and do not collapse with expiration, as is so common with the lax-walled cavities eliciting cavernous breathing. While the pitch of amphoric respiration is slightly higher than that of cavernous, it is lower than bronchial respiration; and further, the pitch of the expiratory sound is a little lower than of the inspiratory. The amphoric quality may accompany both or either of the sounds, but is especially distinct during expiration.

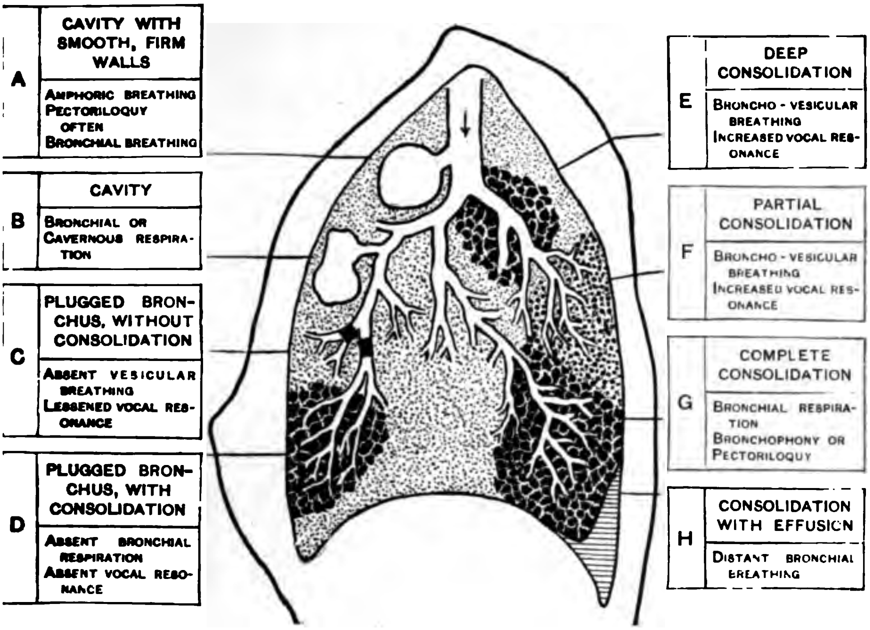

A
CAVITY WITH SMOOTH, FIRM WALLS
AMPHORIC BREATHING
PECTORILOQUY
OFTEN
BRONCHIAL BREATHING
B
CAVITY
BRONCHIAL OR CAVERNOUS RESPIRATION
C
PLUGGED BRONCHUS, WITHOUT CONSOLIDATION
ABSENT VESICULAR BREATHING
LESSENED VOCAL RESONANCE
D
PLUGGED BRONCHUS, WITH CONSOLIDATION
ABSENT BRONCHIAL RESPIRATION
ABSENT VOCAL RESONANCE
E
DEEP CONSOLIDATION
BRONCHO-VESICULAR BREATHING
INCREASED VOCAL RESONANCE
F
PARTIAL CONSOLIDATION
BRONCHO-VESICULAR BREATHING
INCREASED VOCAL RESONANCE
G
COMPLETE CONSOLIDATION
BRONCHIAL RESPIRATION
BRONCHOPHONY OR PECTORILOQUY
H
CONSOLIDATION WITH EFFUSION
DISTANT BRONCHIAL BREATHING

FIG. 40.—SCHEMATIC DIAGRAM OF THE VARIETIES OF BREATHING AND VOCAL RESONANCE IN DISEASE. (Butler.)

Causes.—Amphoric breathing is always pathologic, and due either to a pulmonary or pleural air-cavity communicating with a bronchus, in the latter case producing what is known as *open, circumscribed pneumothorax.*

Although this variety of abnormal respiratory sound is probably heard more frequently as produced by pneumothorax, the conditions of its occurrence in connection with pulmonary excavation will be considered first, because of their physical similarity to cavernous respiration.

(*a*) To give rise to amphoric respiration the cavity in the lung must be of considerable size (of an orange or fist), have tense, firm, nearly uniform walls in thickness and density, smooth upon their interior; have communication with a large bronchus, practically empty of exudate, and superficial in location. It is only in cavities of large dimensions that amphoric echo is produced, whether by the free to-and-fro movement of air in and out of the chamber, or by the factor of *consonance*—that is, the intensification of the normal breath sounds in their transmission through the cavity, which acts as a resonator. Very likely the latter explanation holds in a majority of instances, for the restricted movement of the chest wall corresponding to cavities, as well as to a pneumothorax, would indicate that the entrance and exit of air is hardly forcible enough to develop amphoric breathing. Indeed, in cases of pneumothorax, the affected side may be entirely immobile and the lung tissue collapsed, so that here *amphoric echo* is acquired by the bronchial breathing in its passage through the pleural air-chamber to the surface by means of the fistulous opening in the lung.

Again, unless the walls of the cavity possess a nearly uniform density and thickness and internal smoothness, the sonorous vibrations cannot be reflected with that degree of regularity needful for the formation of a tone really musical in character (Guttmann). Further, the walls must not collapse with expiration, as in cavernous respiration. It must be quite obvious that the communicating bronchial tube must be patulous and of sufficient caliber, and the cavity contain so little liquid that the consonance will not be interfered with. Large cavities usually extend so near the surface that the amphoric sound is loudly audible, especially anteriorly and at the middle height of the thorax, where they are more commonly located, rather than at the base or apex.

(*b*) Amphoric respiration is met with most distinctly in cases of pneumothorax, provided there is a patent pleural opening above the surface of any fluid present, that it communicates with a large bronchial tube, and that the lung is not so completely collapsed that no air can possibly enter through the pleural fistula; in many cases the ruptured pleura heals before total collapse ensues. Here the persistence of amphoric echo is due to the transmitted breath sounds through the lung and pathologic pleural air-chamber.

(4) **Metamorphosing Breathing** (*Seitz*).—This is a modification of bronchial breathing, especially of the inspiratory sound. The first third of inspiration is characteristically harsh, tubal, blowing; this harshness then suddenly changes to a softer bronchial, cavernous, or amphoric breathing, which lasts throughout expiration. Seitz's metamorphosing respiration is met with (rarely) as a sign of cavity of the lung. Its physical explanation resides in the fact that there is a narrowing or stenosis of the bronchial tube, near its entrance into the cavity, which is suddenly overcome by a vigorous inspiration, thus either dilating the orifice or expelling plugging mucus, or both. The change from the bronchial to a cavernous type of breathing may be accompanied by râles, due to the disturbance of liquid in the cavity. A divided inspiration is an inconstant sign of pulmonary cavern; but when repeatedly made out is a distinctive sign.

CHAPTER VII

AUSCULTATION (*Concluded*)

AUSCULTATORY PHENOMENA (Concluded)

SOUNDS OF ABNORMAL RESPIRATORY CONDITIONS (Concluded)

(*B*) *Adventitious Sounds*

Râles.—The sounds produced by the act of breathing may be normal or abnormal. The sounds which are now to be considered are *always abnormal, adventitious,* and not modifications of a natural sound. They are new or superadded sounds, either intrapulmonary or pleural in origin, accompanying, or obscuring, or even supplanting the respiratory sounds.

As soon as the normally smooth mucous membrane becomes swollen and roughened by inflammation, or an abnormal increase in the production and accumulation of secretion takes place, either viscid or fluid, and either in the bronchial tubes or air-cells, adventitious sounds are heard along with the respiratory murmur.

The accessory sounds which are generated within the respiratory passages and lungs are called *râles* or *rhonchi,* the term râle being the one commonly used to designate adventitious sounds produced in the bronchi, alveoli, and cavities of the lung. *Pleuritic friction sounds* are caused by affections of the pleuræ. Unclassified adventitious sounds are the *metallic tinkling* and *succussion* or *splashing sounds.*

Râles (French, denoting a rattling) are divided, according to their quality into *dry* and *moist*; according to the location of their causation they may be laryngeal, tracheal, bronchial, vesicular, or cavernous. *Crepitation* or *crackling* may be dry or moist (*subcrepitant râles*).

The following scheme indicates the varieties of râles usually met with:

RÂLES

(1) *Laryngeal and Tracheal.*	
(*a*) Dry.	Croup, tuberculosis, laryngitis.
(*b*) Moist.	" Death-rattle."
(2) *Bronchial.*	
(*a*) Dry.	
1. *Sonorous*, large or coarse.	Acute bronchitis (larger bronchi and asthma).
2. *Sibilant*, small or fine.	Acute bronchitis (smaller bronchi and bronchioles).
(*b*) Moist.	
1. *Bubbling*, large or coarse *mucous*.	Acute bronchitis (second stage), chronic bronchitis.
2. *Bubbling*, small or fine mucous, or *subcrepitant*.	Capillary bronchitis, edema of the lungs, bronchopneumonitis.
(3) *Vesicular.*	
(*a*) Dry.	
Crepitant, or fine crackle.	First stage of acute lobar pneumonitis, atelectasis.
(*b*) Moist.	
Fine, soft crackle, " mucous click " (same as subcrepitant).	Softening stage of small tubercular deposits in the alveoli.
(4) *Cavernous.*	
Gurgling, large, liquid.	Phthisical cavities partially filled with liquid secretion.

In the writing of Laennec: " Words will often fail me to express their characters, or at least it will be difficult for me to describe them in a manner sufficiently accurate to give a correct idea to him who has never heard them." The terms " dry " and " moist " are used simply to indicate the " impression made on the observer's mind as to whether the sound is produced by dry or moist conditions."

(1) DRY RÂLES (*Rhonchi*).—These sounds are commonly produced by a viscid, scanty exudate in the bronchial tubes, or by a degree of narrowing of their caliber by the inflammatory swelling of a bronchial catarrh. Although there is some moisture present,

the term "dry" is used in a relative sense, as the impression of bubbling given by the moist râles is wanting; the exudate is drier and scantier.

Dry râles are divided according to their pitch, quality, and seat of production into *sonorous* and *sibilant*. They have distinct duration, whereas moist râles are instantaneous.

Sonorous rhonchi are low-pitched, having a snoring, purring, or somewhat groaning character, rather loud in intensity, and produced usually in the larger bronchial tubes. These deep-toned, humming râles may occur as a slight, short, single rhonchus, or may be loud enough to be audible at a distance from the chest, and numerous and persistent throughout the respiratory act; indeed, they may obliterate the breathing sounds. Because of the fact that the tough mucus is frequently so disposed as to practically change the bronchial tubes into wind instruments, the râles produced take on a *musical, consonant*, buzzing quality. Sometimes a very coarse, low-pitched type of sonorous râle, associated with hoarseness and stridulous breathing, may be heard in connection with inflammatory membranous conditions of the larynx, trachea, and main bronchi, and may communicate a distinct fremitus to the hand applied to the chest.

The *conditions* giving rise to these sonorous râles are lessened caliber from tumefaction of the mucous membrane, as in the early stage of bronchitis; the presence of viscid mucus adhering to the swollen, roughened, even—in the very intense acute cases—slightly corrugated surface, and local pressure from without by a tumor or exudation. In the first two instances the rhonchi are heard best over the upper anterior part of the chest, and between the shoulder-blades; in the last, they are circumscribed to the region of the inward-projected bronchial wall. More rarely, the sonorous râle occurs in connection with pulmonary tuberculosis, where the tenacious mucopus may partially plug a large bronchus.

The *sibilant rhonchus* has a characteristic high-pitched, whistling, hissing, squeaking, or shrill piping sound, also decidedly musical, heard pretty much all over the chest, and originating in the smaller bronchial tubes and bronchioles. In the medium-sized tubes the râles may partake of a sort of cooing quality. The intensity is usually less than that of the sonorous râle, but is quite variable. Where there is considerable thickness of exudate coating the mucous lining of a middle-sized bronchial tube, it practically converts it into a small-calibered tube, with the production of true high-pitched sibilant râle. *Musical* whistling tones may be caused by tense threads of ropy mucus stretched across the bronchial lumen, and set into vibration by the respiratory currents of air like the strings of an Eolian harp.

Sibilant râles may be caused by tough, scanty secretion in the fine tubes in capillary bronchitis or bronchopneumonitis before liquid mucus or muco-pus has begun to form, or by the spasmodic muscular constriction of the bronchioles in asthma, in which the characteristic wheezing, whistling, and squeaking noises are produced. They are heard also in the chronic bronchitis of emphysema.

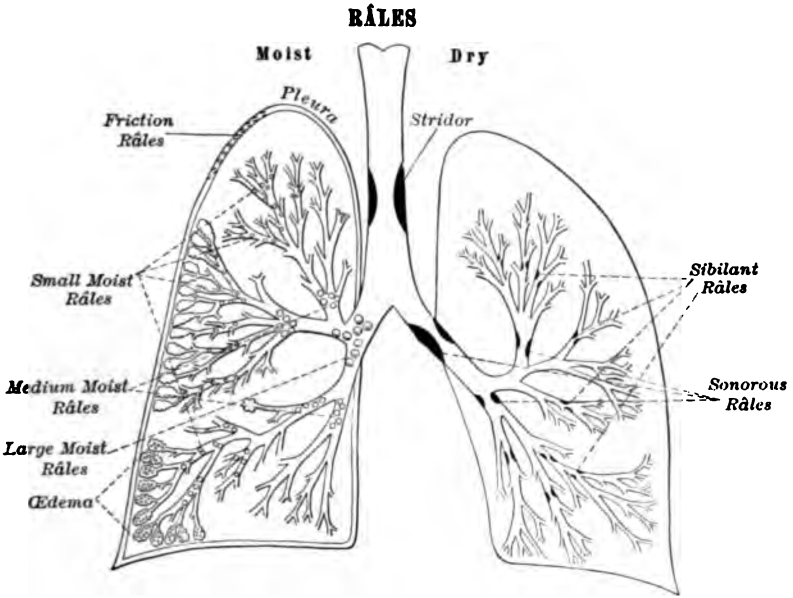


FIG. 41.—DIAGRAMMATIC ILLUSTRATION OF DRY AND MOIST RÂLES. (Le Fevre.)

(2) MOIST OR BUBBLING RÂLES.—As the qualifying names indicate, these râles have a quality of soft, bubble-forming or -bursting, arising principally from the presence of more or less fluid exudate in the bronchial tubes. Moist râles are divided into *large*, *medium*, and *small*, according to the apparent size of the bubbles, or of the bronchial or pulmonary cavities in which they are produced. Their intensity is variable, the larger the bubblings the louder, with equal force of breathing. The pitch, also, varies according to the size of the râles, and somewhat upon the degree of fluidity of the disturbed substance; those produced in the main bronchi, for example, by the vibrations of thin, watery mucus, serum, blood, or pus are decidedly lower in pitch than those set up in the medium- or small-sized tubes by the movements of air through a thicker, stickier liquid.

Gurgles or *gurgling râles* are the loudest, lowest-pitched râles usually met with. They may be tracheal or cavernous in their seat of origin. The *tracheal* râles are the coarse bubbles which occur in connection with many terminal conditions, where the rapidly failing powers and comatose state allow usually the seromucous liquid from the filling, edematous lungs to gradually creep up to the tracheal level, thus constituting the so-called "death-rattle," although death does not always and necessarily ensue when these large tracheal gurgles are heard. They are distinctly audible without the aid of a stethoscope, and often at a distance of several yards from the patient.

Very diagnostic, however, are the large liquid gurgles of hollow, metallic quality heard over phthisical cavities of the lung, where there is a considerable amount of fluid, and especially when communicating with a bronchus dipping below the level of the latter. Gurgling râles are heard about equally well during inspiration and expiration, and are also excited readily by coughing. The presence of a large bubbling or cavernous râle at either apex cannot arise from the bronchial tubes within, as they are too small, but must mean a large pathologic space—a cavity, especially if the râle seems close to the auscultating ear. Elsewhere, large or medium moist râles may indicate also bronchial dilation, and have a more distant quality.

Cavernous gurgles may be absent, constantly or temporarily, because of the following conditions: The cavity may be empty of liquid; it may be entirely filled with liquid, excluding the air; the level of the liquid may be below the opening of the bronchial tube; the latter may be obstructed, as by a plug of inspissated muco-pus; rarely, the cavity may be partially or completely occluded by the pressure of a pleuritic effusion.

Large mucous râles, of a loud, low-pitched, bubbling character—though lacking the hollow, deep sound of the cavernous râle—when heard over the position of the main bronchi, are significant of the stage of free liquid secretion of acute or chronic bronchitis. Similarly, slightly higher-pitched, medium-sized moist râles, heard lower down and in the interscapular region, over tubes of moderate caliber, indicate the presence of a deeper-seated bronchitis with mucoserous or purulent fluid, or of a hemorrhage into the tubes, or, possibly, of pulmonary edema, the transuded liquid having risen to the level of the tubes.

The *fine moist* or *subcrepitant* râle is the smallest of the râles having a moist quality. It is produced in the finest tubes or bronchioles. Its character is that of the bursting of the tiniest bubbles, or, more often, the separation of the moist, sticky walls at the time of the respiratory passage of air-currents. This râle is so fine and high-pitched that it approaches the dry, crackling quality of the crepitant râle (*vide*); hence the term, *subcrepitant*. While, as just intimated, this fine moist râle may arise from the air-disturbed liquid secretion in the bronchioles or air-cells, the more probable explanation is that it depends upon the rapid and sharp "separation of the agglutinated walls of these finer air-passages from each other" rather than upon the formation and bursting of bubbles. The force of respiration is so slight, and the caliber of the tubes is so small, as to be insufficient for the raising of bubbles. Because of the greater force of inspiration, therefore, the subcrepitant râle is usually more evident during, or rather near, the end of the inspiratory breath sound, but also with the expiratory sound if the exudate in the bronchioles is not too tenacious. A sound similar to the finest moist râle, as produced in the terminal bronchial tubes and vesicles, may be obtained by pulling apart the thumb and finger or palms of the hands, which have previously been wetted with a sirupy or gummy liquid.

The *conditions* causing the subcrepitant râle are numerous. They may be local or more or less general. When present only in one apex, a number of these small, moist, crackling râles indicate beginning tuberculous bronchopneumonia—phthisis, indeed, as a single "click" heard over an apex is almost diagnostic of incipient tuberculosis; but they may be heard also during the second stage of the disease, while cheesy degeneration and liquefaction of the tuberculous deposit is going on.

They may be detected around the borders of pneumonitic consolidation from various causes, on account of the adjacent collateral edema or congestion, or bronchial catarrh. As accompaniments of the so-called lobular, catarrhal, or bronchopneumonitis of the very old, and of young children, they are characteristic; here they are due to the capillary bronchitis in the second, or moist, stage, the subcrepitant râles following the sibilant rhonchi of the first, or dry, stage. In this disease the râles occur over multiple small areas posteriorly, or, in extensive cases of bronchiolitis, may be heard all over the chest.

As the so-called *râle redux*, the subcrepitant râle is commonly present in the later or resolving stage of lobar pneumonitis.

The discovery of fine moist râles in the infrascapular regions may point to pulmonary edema, hypostatic congestion, or an effusion of blood into the bronchial tubes.

Variations and *conditions modifying* the *bronchial râles* are important for consideration before taking up the crepitant râle, which, when not pleuritic in origin, is produced abnormally within the alveoli.

Besides the variations in quality and pitch already given, the size, loudness, and location of the râles, their bronchial origin is characterized by the facts that they are most changeable as to audibility or intensity in any particular locality; also as to number and the effects of cough. The additional significance that the bronchial râles accompany both inspiratory and expiratory breath sounds, and that their tone or character is influenced by the condition of the surrounding lung, must also be noted.

Further details of importance in diagnosis are appropriately considered, therefore, at this juncture.

In the first place, râles vary greatly in *character*. This, as previously intimated, is a feature of the bronchial râles, and reference to it bears repetition here in a more specific way. During and after an asthmatic paroxysm, for instance, a multitude of all sorts of râles may be heard within a few hours. In a case of bronchitis, dry râles are commonly followed by moist râles. At one time the râles may be large and bubbling, and shortly afterward these may be replaced by small moist râles as the pathologic condition extends downward.

Secondly, bronchial râles in particular exhibit differences in *intensity*. Their loudness, however, does not depend upon their number, but upon the force of the breathing. The value of the attribute of loudness rests upon the localization of the râles with more precision; the louder a râle becomes in moving the ear over the chest the nearer one approaches the spot of its generation. This involves the question of the *transmission* of râles. It should be remembered that the region where a râle is heard at once does not necessarily correspond to the part of its origin. It is rare, however, to find râles transmitted from one lung to the other; that is, if râles are audible over both sides of the chest, it may be inferred that the cause exists bilaterally, as in bronchial asthma. Usually the dry râles are conducted to a greater distance than even the loudest of the bubbling sounds. When the adjacent lung tissue is thickened from any cause, increasing its conducting power, the râles produced beneath are heard with exaggerated intensity. In cases having very loud, especially sonorous rhonchi, and where the thoracic walls are comparatively thin and elastic, their vibrations are transmitted to the surface so as to be palpable—rhonchal fremitus (see under Palpation).

Thirdly, the *irregularity* and *inconstancy* of the bronchial râles is very characteristic. This applies especially to their evidence and recurrence, as influenced by coughing and breathing. Thus they vary as to *location.* Before one may be assured that there are no râles present in a patient, it is always well to have him cough, and then take a full breath a few times. Both dry or musical and moist or bubbling râles may be developed or increased, diminished or removed at a given spot by cough or forced respiration. They may occur at several different places within a short time, in the most transient, fugitive manner; appearing where before they were absent, disappearing where they were present, shifting from place to place from day to day, or even while the patient is under examination.

Fourthly, the *stage of respiration* in which the râles occur is quite significant in most cases. Bronchial râles, dry and moist, may be heard during both inspiration and expiration, or either alone. The sibilant rhonchi are more frequently audible toward the end of inspiration, except in asthma and emphysema, where the expiratory difficulty causes exaggeration of the force of that act by the auxiliary muscles of respiration. The sonorous rhonchi may be heard throughout the whole of inspiration and expiration, provided the latter is not unduly weakened. The large moist râles are also nearly continuous, especially in diffuse bronchitis with copious liquid secretion. If scanty, the bubbling sounds may be limited to the inspiratory portion of the breathing. Sometimes the râles occupy the whole of the respiratory act so completely and loudly that the breath sounds may be entirely obscured. The subcrepitant râle is not, as a rule, evolved with a promptness simultaneous with the beginning of inspiration, but is apt to occur with variable suddenness at the end of inspiration or the beginning of expiration. The râles are few in number, and often dissimilar to each other. They are influenced by coughing, and manifest the changeableness of other bronchial râles.

Fifthly, bronchial râles may be modified in quality according to the *condition* of the *adjacent lung.* They may have a *resonant* or *ringing,* even *metallic* character. Thus the dry, humming, purring, sonorous râles may change from a toneless noise to a clear, resonant, almost musical sound. This happens in certain cases of bronchitis with tough mucus accompanied with thickening of the lung, as at the beginning of the second stage of pneumonia. Sibilant rhonchi are seldom "consonant" or ringing, owing to the damping effect of the surrounding air-filled vesicles in the cases of asthma and emphysema in which they are commonly heard. More often moist râles become ringing.

Besides *consolidated lung tissue*, resonant râles arise in close proximity to *cavities*. The ringing râles bear about the same acoustic relation to the non-ringing that bronchial breathing does to normal vesicular breathing. The explanation is found in the fact that dense tissue transmits the râle sound with a favorable intensification. If the individual bubbles are few and of deep-seated origin, their resonant character may be barely noticeable. Further, it is not invariably true that moist bronchial râles within the neighborhood of condensed tissue are ringing in quality; for the area of thickening may be small, or there may still be patches of expansible, spongy lung tissue between those of the deposits of thickening.

The clearest ringing moist râles occur within or very closely to superficially situated cavities of fair or large size, and surrounded by a zone of consolidated lung parenchyma. Here the ringing may assume a distinct clanging (*klang*), or metallic tone, thus corresponding to amphoric resonance and breathing as the ordinary ringing râles do to bronchial breathing.

While the *resonant* râles, then, indicate the positive presence of consolidation or excavation of the vesicular tissue (except as below), on the other hand, non-resonant râles do not necessarily exclude such conditions. In instances of the latter, the physical changes are detected by dulness or tympany, as the case may be, and, on auscultation, bronchial or cavernous respiration.

With two exceptions, ringing râles are practically never associated with vesicular breathing. In the first place, in the simple bronchitis of children, without any evidence of consolidation or cavity, the râles not infrequently have a resonant character because of the marked elasticity of the lungs and chest wall. Again, in certain cases of emphysema, along with the generalized sibilant and snoring rhonchi, there may be a circumscribed region over a lower lobe, without bronchial breathing and dull percussion note, that is, evidence of condensation of lung; or of tympany and cavernous or amphoric breathing, that is, signs of cavity; and yet the presence of ringing râles: here we may infer a deep-seated *bronchiectatic cavity*, or possibly a bronchopneumonic deposit, to account for the phenomena.

(3) THE CREPITANT RÂLE.—This râle is distinctive because it is the finest of râle sounds, having a special acoustic quality which

permits it to be classified as the finest moist sound as regards its origin, but is more often referred to as a dry râle because of its peculiar quality; the quantity of moisture is so small, and the spaces within which the crepitations are produced are also so small—namely, the alveoli and terminal bronchioles—that the sounds consist of a number of fine, dry *crackles*; hence the term. The fineness and dryness of the râle may be imitated by rubbing a small lock of hair between the finger and thumb close to the ear, or by throwing salt on a fire. The crackles are entirely wanting in the fine, moist, almost bubbling quality of the subcrepitant râle. Other *points of differentiation* between these somewhat similar adventitious sounds are as follows: (*a*) The crepitant râles are more numerous than the subcrepitant, the minute crackles succeeding each other so rapidly that to the tyro's ear they may seem like a continuous sound—like the very distant, hardly audible explosion of a bunch of firecrackers; (*b*) crepitations are uniform or equal in size, while the subcrepitant râles are variable in size; (*c*) the crepitant râles are not influenced by coughing, as are the subcrepitant, being heard equally well, if not better, after as before the cough, probably because of the temporary increase in the fulness of the inspirations.

Until recently it was taught that the crepitant râle occurred invariably and pathognomonically at the time of inspiration, and usually at the end of inspiration. While this is true in the majority of cases in which the crepitant râle is met with, nevertheless it is often heard at the beginning of expiration, and, at any rate, the precise time at which it is heard depends upon the pathologic condition causing it. Thus, it is generally spoken of as the characteristic râle of the first or congestive stage of acute lobar pneumonia, in which it is held that the pulmonary capillaries of the affected lobe, soggy with an excess of blood, permit an incipient transudation of some of the latter's serum, causing an agglutination of the intravesicular walls with the expiratory contact, the separation of which with the succeeding inspiration, however, giving rise to a number of fine crepitations corresponding to the number of vesicles (or terminal bronchioles) in which the sticky contact is overcome. This explanation makes the crepitant râle exclusively intrapulmonary in origin.

On the contrary, the extrapulmonary or pleuritic causation of the râle has its advocates also. That is, it is maintained that it is nothing more than the finest kind of pleuritic friction sound. For it is a well-known fact that the pleural covering of that portion of the lung affected by the pneumonitic process is practically always in-

neously, and really produces the "stitch in the side"
plained of by pneumonia patients. Undoubtedly, the
nt of the viscid pulmonary and costal pleural surfaces at
end of spiration, and their separation or slight friction at the
of expiration, are frequent contributory causes in the pro-
ction crepitant râles. In a certain number of cases I have
n led to infer that the regular, persistent occurrence of crepita-
at the end of inspiration only, disappearing with the positive
lishment of consolidation twenty-four hours later, pointed to
r intrapulmonary origin. Again, their coincidence with the be-
ing of expiration, as accurately determined, and their continu-
e when the vesicles w filled firmly with fibrinous
date, along with th being produced near the sur-
e, was equally signifi causation. So that, as usually
nspires, the truth embraces s, as to credibility and proba-
bility. A third explanation, that tant râles are "caused by the
bursting of fine bubbles formed by forcing out during inspiration
of the secretions from the br s into their infundibuli," is
more consistent with the deve of patches of bronchopneu-
monitis than of lobar pneumo ral crepitations may be diag-
nosticated sometimes by eliciting a ater abundance of them, with
increased loudness, by the pressure of the stethoscope with persistence after coughing.

The crepitant râle is sometimes heard accompanying the subcrepitant in the final stages of resolution in pneumonia (*crepitans redux*). It is present, also, in connection with cases of *tuberculosis* (apices), *catarrhal* or *bronchopneumonitis* (bases), *infarctions*, *atelectasis*, and *edema of the lungs*, although more rarely than the finest bubbling or subcrepitant râle.

Atelectatic crepitations deserve brief separate consideration. They may be intimately connected with pathologic pulmonary conditions, or only remotely so, as on the border-line of the normal. As regards the former, they may occur over lung that is partially compressed by a pleural effusion, and the patient is induced to take deep inspirations while one listens; or over collapsed lung, similarly, when either main bronchus is compressed—by a large aneurism, for example—or occluded within by a foreign body. Patches of atelectasis between areas of bronchopneumonia posteriorly may be manifested by scattered crepitations, when other physical signs are absent or elicited dubiously, on account of the smallness of the areas. Here one must, furthermore, be guarded against mistaking the subcrepitant râle which accompanies the bronchiolitic catarrh for the finer, drier, higher-pitched crepitant râle.

Again, almost *physiologic* are the crepitations heard at the end of a deep inspiratory effort in those who are congenitally, habitually, or because weak, sickly, or invalided from other diseases, poor breathers. Those, particularly, who are predisposed to "consumption" seldom inflate the apices of their lungs with ordinary respiration, and do so with difficulty, even by forcible breathing, as they may be able. Patients who are obliged to remain in bed because of protracted, exhausting illness, not infrequently give evidence of vesicular collapse by the inspiratory crepitations heard with increased respiratory efforts; these are usually found at the base of the lungs, at their borders, laterally and posteriorly. In fact, in many persons of ordinary health, but whose respiratory function is superficial and sluggish, as in those with sedentary habits, or who, in standing or sitting, habitually assume faulty, confined, stooping, round-shouldered attitudes, fine crepitant râles are often heard along with the full breathing needed to overcome the atelectasis. We discover them over the apices of the young—students, clerks, factory and shop hands, etc.; quite commonly at the bases, especially in the lower axillary regions, of individuals over forty or forty-five years of age—as in professional and business and club men, government officials, and so on, who sit and ride too much, and walk or exercise otherwise too little out-of-doors. Such crepitations usually disappear after four or five full breaths have been taken.

Laryngeal crepitation, as a *sign* of *tuberculosis* (Cybulski: confirmed by Remouchamps, *La Semaine médicale*, No. 48, p. 392, December 2, 1903), I can also testify to as of distinct confirmatory value when present, although its absence does not preclude the possibility of the disease being present, nevertheless. It may, however, in occasional cases of incipient tuberculosis, serve to determine the diagnosis when other auscultatory phenomena fail to give positive results.

The method is simply to have the patient preferably lying, with the mouth slightly open, while the physician listens with the ear at a distance of from 5 to 10 cm. from the patient's mouth. If the individual is affected with pulmonary tuberculosis, a fine crepitation is heard in many cases which is termed laryngeal, because its maximum intensity is near the larynx. The sound resembles the scratching of a pen moved softly over paper. It is heard both during inspiration and expiration, but, in my experience, is usually more marked during the latter. This laryngeal crepitation persists during the whole course of the disease, and this persistence, together with its increase or diminution along with the aggravation or amelioration of the malady, and the absence of the sonorous and sibilant, or mucous râles noted in bronchitis, constitute the chief points in establishing the diagnosis.

The auscultator must guard against being deceived by *artificial crepitation*. This sound is produced, so loudly at times as to interfere seriously with the perception of the respiratory sounds, by the rubbing of hairs on the chest against the end of the stethoscope. This source of error, as pointed out before (see Method), may be eliminated by simply wetting or oiling the hair until its crispness is overcome, and it lies flatly and closely to the skin.

The "*mucous click*" is a fine, soft, moist crackle or "click," usually occurring singly, and not removed by coughing (Loomis); it is virtually an isolated subcrepitant râle. The term having been early associated specifically with the diagnosis of incipient apical tuberculosis, has persisted hitherto in clinical language in the same connection. It is probably due to the forcible and sudden inspiratory separation of the walls of a small bronchus which have been adherent because of the viscid secretion of a tuberculous bronchitis, or of the softening down of an adjacent lobule of tuberculous deposit.

In general, it may be said that in the majority of instances where râles are heard their origin is bronchial; that crackling râles, whether dry or moist, occurring at the bases of the lungs, usually indicate some bronchopulmonary congestion there (pneumonitic); at the apices, usually a tuberculous broncholobular inflammation or infiltration.

Pleural Adventitious Sounds.—PLEURITIC FRICTION SOUNDS.—In health, the smooth, serous, thin pulmonary and costal pleuræ glide over each other during the respiratory movements without noise. Any diseased condition of the pleural surfaces, therefore, which produces abnormal roughness, dryness, or thickness is usually accompanied with some kind and degree of friction or rubbing sound.

The favorite *location* of pleuritic friction is over the lower zone of the chest, laterally and posteriorly, but especially in the axillary region, where the excursion of lung movement is greatest, and costodiaphragmatic pleural apposition is closest. The friction sounds may be heard over a large area at the base of the lung, on one side only, as a rule, as in ordinary pleuritis, or rarely it may be localized over a small area at the apex in early tuberculosis (dry tuberculous pleurisy). Again, quite commonly, it may occur in small patches, one or more, in the axillary and infrascapular regions.

The *quality* and *intensity* of the pleuritic friction sounds vary considerably according to the character, chronicity, and degree of the pathologic changes. When the sounds have a rough rubbing, grazing, shuffling, grating, rasping, or even creaking or "leathery" character, the pleural surfaces are manifestly dry and roughened by a fibrinous, cohesive exudate (*pleuritis sicca*). At the onset of a pleuritis, when the only change is the early dryness of the congestion of the membrane, the friction sound is hardly more than a slight, single, abrupt, grazing or soft scratching noise, audible over a variable, but usually limited, extent of surface. Moderately and extremely loud rubbing, grating, or scraping sounds point to increased intensity of pleural inflammation and quantity of plastic exudation upon the apposed surfaces, with consequent aggravated attrition.

As suggested by Cabot, "the sound of pleural friction may be closely imitated by holding the thumb and forefinger close to the ear, and rubbing them past each other with strong pressure, or by pressing the palm of one hand over the ear and rubbing upon the back of this hand with the fingers of the other." Friction sounds seem very near to the ear, and their superficial character may be attested by noting their intensification when external pressure is made against the chest with the head in immediate auscultation, or with the Bowles stethoscope. The loudness of intrapulmonary râles cannot be affected so. Not infrequently, in marked cases associated with a harsh grating sound, the friction may also be palpable (fremitus), and equally sensible to the patient as well.

In *pitch*, the pleuritic friction sounds vary greatly, but, as a rule, they are distinctly higher pitched than the adjacent respiratory sounds.

The *duration* of the individual frictions depends mostly upon the extent and continuity of the pleuritic sticky exudate, the dilatability and freedom of motion of the lung, and upon the amount of pain. A single short rub, or several short, scratchy sounds following each other in rapid succession without the intervention of a pause, signifies one or more small patches of circumscribed pleuritis, often bunched together. Again, even in well-marked cases of extensive plastic inflammation of the pleuræ, the stitchlike accompanying pain causes what might otherwise be a friction sound of long duration, merely the shortest, catchy, interrupted grazing. Directly connected with this element of duration is the relation of friction sounds to the respiratory *rhythm*. They usually accompany both inspiration and expiration—to-and-fro sound; frequently they are heard only during inspiration alone, and only rarely do they exist with expiration alone. They are louder during inspiration, for obvious reasons, and are most audible at the end of this act, particularly the lighter rubbing sounds. The loud rasping and creaking frictions of cases of pleuritic thickening and roughening in the chronic pleurites, sometimes with calcareous infiltration in very old people, may be heard throughout the greater part of both inspiration and expiration. Disappearance of the friction sounds after continued deep breathing for a minute or two is explained by the smoothing out of the rough elevations causing the friction, just as one might separate the buttered surfaces of two slices of bread put together—first by lifting or pulling them apart, thus showing the irregular, wavy appearance, and then, after pressing them together again, remove them past each other by a steady, gliding motion, thus leaving the buttered surfaces almost as smooth as when spread with a knife.

For the purpose of eliciting a friction sound that may have disappeared, or when one is in doubt as to the presence of pleuritic friction at all, the *arm* or *decubital maneuvers*, as suggested by Abrams, may be resorted to. The first consists in having the patient suspend respiration while he or the physician raises the arm on the affected side over the head; at the same time the suspected area is auscultated. This maneuver reverses the direction of the movement of the parietal against the pulmonary pleura, and thus may be the means of eliciting a slight pleuritic friction that may have disappeared because of a previous smoothing out of roughnesses in the ordinary act of respiration. The decubital maneuver is described as follows: "Let the patient lie upon the affected side for a minute or two, then let him rise quickly and suspend respiration. Now listen over the affected area, at the same time directing the patient to take a deep breath."

The friction sound is changed very little, if at all, by coughing, and may be increased slightly after that act.

When a pleuritic friction sound ceases to be heard, quite often it means that the surfaces of the pleuræ have become separated by the production of a liquid effusion, although, on the other hand, the presence of such an effusion does not invariably preclude the audibility of a friction sound being heard even over the seat of the effusion, as in occasional cases, particularly in children, the sound produced above, where the pleural surfaces are still in contact, may be conducted downward along the chest wall. As the fluid becomes absorbed, or removed by tapping, in some cases the renewed contact of the thickened pleural surfaces produces a coarse friction sound often louder even than during the first stage of the pleuritis. The persistence of this phenomenon may be sensible to the patient for years after recovery, and may cause considerable anxiety unless it is explained that pathologic or structural changes do not regain the normal, although clinically or functionally he may be quite well; just as a scar, while it represents a healed wound, is nevertheless a permanent anatomical alteration.

Pleuritic crackling or *crepitation* consists of the finest friction sounds, closely simulating the crepitant râle of pneumonia; indeed, as pointed out in describing that râle, some clinicians assume its pleuritic origin exclusively. Its differentiation has been indicated, and will be referred to further, included as a pleural sound, in the next paragraph but one. Here should be mentioned the very fine, soft, merest *rustling* sometimes heard around the sides and back of the base of the chest in *acute miliary tuberculosis*, the sound being produced probably by the rubbing of the numerous tiny tubercles studding the pleural membranes, and projecting slightly above their surfaces (*subpleural friction*).

Conditions other than simple acute plastic pleuritis, or chronic pleuritis, but which are revealed in part by the friction sounds of the accompanying dry pleuritis, are as follows: Consolidation of the lung from tuberculosis or pneumonitis (already referred to), *pneumonokoniosis*, *bronchiectasis* with *reactive pneumonia*, *pyemic deposits* in the *lungs*, *infarction*, and *emphysema* (Vierordt). Friction sounds of a distant character heard at the base of the chest, just below the lung borders, are usually significant of *diaphragmatic pleuritis*, a condition very painful, often alarming in its immediate symptoms, and easily overlooked because of its anatomic proximity to such organs as the heart, liver, and stomach, to which the clinical manifestations seem more commonly and readily referable. Friction sounds detected at the base of the right lung posteriorly, or in the seventh, eighth, or ninth right interspaces anterolaterally, may be due to a perihepatitis from hepatic abscess or cancer, or to a subdiaphragmatic (subphrenic) abscess, and must be differentiated and localized carefully, especially when affected by the respiratory rhythm, from those of true diaphragmatic pleurisy.

The principal points of differentiation between the pleuritic friction sounds and the bronchial râles it is important to apprehend, as bronchitis is frequently associated with pleuritis. They may be summarized and tabulated as below (modified after Le Fevre):

Bronchial	*Pleural*
Not generally localized, but if so, they are associated with signs of consolidation.	Strictly localized.
Usually bilateral, and variable as to location, front or back, above or below.	Unilateral, constant, and usually at the base.
Seldom accompanied by pain.	Characteristic pain frequent.
Various qualities of dry and moist râles, remote from the auscultating ear, and unaffected by external pressure.	Different qualities, but all giving impression of friction or rubbing, near to the ear, and intensified by pressure over the pleuritic area.
Location, loudness, number, and size of râles variously modified by cough and deep respiration.	Are not removed or perceptibly modified by cough; may be temporarily decreased or abolished by forced respiration.
Râle sounds are very rarely palpable.	The friction fremitus frequently accompanies the auscultated sound.

In certain cases of diffuse bronchial catarrh associated with pleurisy, with harshness of the respiratory murmur and bronchial râles, the circumscribed friction sounds existing in the same part of the chest may be detected only with the greatest difficulty, unless they happen to be of the shuffling, creaking, or rumbling character. Repeated auscultation, however, with a discriminating consideration of the phenomena just paralleled, will usually serve to discover the association.

Again, the pleuritic element of a severe case of pleuropneumonia may go unnoticed, because of the greatly restricted movement of the consolidated lung, and consequent lack of audible friction other than some pleuritic crepitation, perhaps.

Pleuropericardial friction denotes the sound produced by the rubbing of the inflamed, viscid pleural surface of the lung surrounding the heart against that covering the pericardium. The rubbing is generally heard only during inspiration, and at the time of the cardiac systole; but if, as in a minority of cases, it is faintly audible during expiration, the sound is intensified, nevertheless, during inspiration. This sound must not be confused with the soft, synchronous cardiorespiratory murmur (*vide*) heard in some normal individuals.

METALLIC TINKLING.—This term was used by Laennec to designate the silvery, fine, resonant metallic sound of single bursting bubbles, or of falling drops occurring in pneumothoracic or large pulmonary cavities partly filled with air and partly filled with fluid. The tinkle has been likened to the sound produced by the dropping of water in a cistern, or of a grain of sand into a large, hollow metallic or glass globe. The intensity is slight, but distinct. The pitch of the tinkle is high, but one may perceive occasionally a closely following deeper tone of resonance—a sort of secondary, reverberating wave. As to rhythm, metallic tinkling may coincide with either or both inspiration and expiration; very often a single tinkle is heard with each phase of the breathing. It may be heard with quiet respiration, or it may be developed only after coughing.

While the conditions for the production of metallic tinkle are principally those found in *hydro-* or *pyopneumothorax*, where one hears the resonant impact of a drop of liquid from the relaxed or collapsed lung, with its exposed cavity or bronchial tube, upon the surface of the liquid at the bottom of the pleural sac, the contained air giving the metallic consonance to the sound, this physical sign may be associated with such other signs of large phthisical cavities, thin- and tense-walled, as marked tympanitic sound on percussion, and amphoric breathing. In some cases it is probable that the tinkle is the result of the bursting of fine, much-inflated moist râles in the tissues surrounding a cavity, the air-chamber of which acts as a resonator.

Finally, it should be noted that a metallic tinkling sound is sometimes heard *normally* over the dome of a gas- or air-inflated stomach containing liquid, especially in the left lower regions of the chest, anterolaterally.

SUCCUSSION OR SPLASHING SOUND.—That this may be heard over the stomach is a common observation, and that it may not be mistaken for a splashing sound produced in the left pleural sac needs but to be mentioned. Its recognition is easy when the conditions of its occurrence obtain; these are, the simultaneous presence of air and liquid in the pleural cavity—that is, a *pyo-* or *hydropneumothorax*. The splash may be heard sometimes by the patient, and felt also, when the body is given a sharp shake sidewise. The physician may shake the patient's trunk gently himself while auscultating the chest, or the sound may be heard at a distance of several feet while the patient agitates the fluid by a bodily jerk. Very rarely the sign may be due to the existence of an enormous pulmonary excavation. Rhythmic (cardiac) splashing sounds may also be heard occasionally after certain stab wounds, because of the presence of air and liquid in the pericardial sac (pneumohydropericardium).

The pitch and intensity (aside from the effect of the violence of the agitation upon the latter) of the succussion sound depend upon the density of the liquid and the proportional quantities of liquid and air present, just as such changes may be perceived on shaking a decanter or jug partly filled with air and fluid. It is important to remember that splashing sounds do not occur in ordinary pleuritic effusion or hydrothorax because of the absence of air. Metallic tinkling may be an associated phenomenon.

The disappearance of the succussion splash in a given case may indicate one of several things: (*a*) Thickness and proportional excess of a purulent exudate—as was mentioned by Hippocrates in describing this sign (Hippocratic Succussion); (*b*) the increase of the pleuritic exudation displacing the air; (*c*) the encysting and circumscribing of the pyopneumothorax by the formation of adhesions.

Water-Whistling, or the Lung-Fistula Sound.—According to Unverricht, who first observed this sign while puncturing and aspirating a case of hydropneumothorax, this consists of a fine metallic gurgling, bubbling, or splashing sound, heard in cases of open pneumothorax where the opening in the pleura is directly below the surface of the liquid. The sound may be simulated somewhat by the blowing of bubbles, or in gargling the throat. Its occurrence and persistence depend upon the position of the patient, and the increase or diminution in the quantity of fluid.

Veiled Puff.—This is a short, hollow, puffing or whiffing sound, sometimes high-pitched, occasionally audible over, and thus diagnostic of, small sacculated bronchiectatic cavities; it is heard at the ending of the inspiratory murmur.

Post-Tussive Suction Sound.—Corwin describes this as a "sucking or sometimes semisonorous sound, which has been heard after cough, in case of cavity with yielding walls and an opening into a bronchus. It occurs with the inspiratory entrance into the cavity of air which has been driven out by compression in the act of coughing."

Indeterminate Râles.—Besides the various dry rhonchi, moist râles, and friction and other pleural sounds, there occur at times certain adventitious sounds which, because of their indefinite, obscure, irregular characteristics, must be classed under the collective term, *indeterminate râles.* Skoda so designated that group of medium-sized and coarse râles indicative of the presence of fluid in some of the larger air-passages, but not of the permeability of the adjacent or surrounding lung tissue; that is, these râles, considered by themselves, signified a bronchial catarrh; but as to whether this was primary and simple, or secondary and complicated by parenchymal consolidation or excavation, was indeterminate.

More particularly, to-day these indefinite sounds include all *crumpling, fine crackling, soft rumbling* sounds, moist or dry, inspiratory or expiratory, or both, which may be of doubtful origin or mode of production or conduction.

They are not necessarily abnormal, or always referable to the respiratory organs. On the contrary, they may be really muscular or integumental sounds in a healthy-lunged individual. For instance, sounds generated by the movements of the thoracic framework in deep breathing may simulate intrathoracic râles. In very old people we sometimes hear creaking cartilaginous sounds (dryness?) that may imitate quite closely the creaking pleuritic friction sound. Again, as noted by Walshe, the fine crepitus of a broken rib may be mistaken for the crepitant râle of a pneumonia. A moist rhonchoid sound, or pseudo-friction noise, may also result from the presence of a dropsical serum in the chest walls. The imitation may be detected by pressing aside the fluid with a stethoscope; by its existing in the abdominal as well as the thoracic walls; by its limitation to edematous portions, and the fact that it is not synchronous with the respiratory movements.

Probably the most distinctive of the so-called indeterminate râles, however, are the *crumpling* or soft rubbing sounds heard at the end of inspiration in emphysema, especially the interlobular variety. The dull, soft crackle has been likened to that produced in bending parchment to and fro.

Crepitations are sometimes heard, variable as to dryness, largeness, abundance, and rhythm, over the sternum, during forced respiration only. They may be physiologic, due to the sudden dilation of the marginal lung vesicles or to fine mediastinal adhesions.

The muscle sounds arising from the constrained posture of the patient, or when the breath is held, have been referred to previously as imitative of intrathoracic râles.

(C) *The Vocal Resonance*

The *auscultation of the voice* is much less important and useful than of the breath and adventitious sounds, and to many physicians whose sense of touch has been at all well trained in palpation, the changes in vocal fremitus are felt with keener perception and surer judgment as to the underlying physical conditions than are those of the vocal resonance. The signs of the latter are more uncertain, confusing, and obscure, and yet they may have occasionally decided auxiliary clinical signification. There is much room for a more extended and discriminating study and analysis, and more frequent and patient practise of this method of auscultation.

Just as in the palpation of the vocal fremitus, so in the auscultation of the *whispered* or *spoken* voice, for the purpose of obtaining information as to the condition of the lungs and pleuræ, the basis of the procedure is found in the fact that the glottic vibrations are transmitted downward through the respiratory tract as well as upward through the mouth, and that they are audibly as well as palpably conducted through the chest wall.

Consequently, also, the degrees of audibleness and character of the vocal resonance are affected similarly to the vocal fremitus by the same physical conditions and changes precisely.

The same guiding rules for auscultating the breath sounds are applicable here. That is, in brief, whether practising immediate or stethoscopic auscultation, the method of rigidly comparing corresponding parts on both sides of the thorax should be adhered to. The ear and stethoscope should not be applied too lightly, to avoid a tremulous character of the resonance; nor, on the other hand, too heavily, that its real and full volume and distinctness may not be artificially diminished. It is essential to have the patient repeat monotonously and evenly the same word or syllable, maintaining the same tone, intensity, and pitch of voice, while different regions of the chest are being examined, so as to insure uniformity of results, as in the performance of palpation of the vocal fremitus.

Normal Voice Sounds.—There are several varieties of vocal or whispered resonance, named after their regions of production in health. Thus, when we place a stethoscope over the larynx or trachea while the person speaks or whispers, we hear the NORMAL LARYNGOPHONY or TRACHEOPHONY. When the patient utters "nine, nine, nine" or "one, one, one," with the natural speaking voice, the sounds are transmitted to the ear of the auscultator with marked intensity and concentration of force, although the words are not perfectly articulated. In persons of loud and grave voice, the vibrations may be almost painful to the examiner's ear. At the upper part of the sternum, over the main bronchi, nearly as far as the parasternal lines in the infraclavicular spaces, and in the upper part of the interscapular region, especially to the right of the vertebral column, the vocal sound is less intense and more confused, so that it is rarely possible to identify the articulate words when the free ear is closed. This is the NORMAL BRONCHOPHONY. It corresponds with the bronchial breath sound, and wherever bronchial breathing occurs pathologically bronchophony is similarly associated. The sound does not seem to smite so directly upon the ear, being more diffused because of its greater distance from the glottis, and the consequent diminution of intensity from the interference of more reflected, reverberating sound-waves. The transmission of the laryngeal, tracheal, and bronchial vocal sounds is accompanied by a *fremitus* or *thrill* perceptible to the examiner's ear, a sign which is absent when the whispered voice is listened to.

The *normal laryngeal, tracheal,* and *bronchial whisper* (*whisper bronchophony*) is heard with a regional intensity analogous to that of the spoken voice. The whispered voice sounds like, as in fact it is, an exaggerated or bronchial expiratory breath sound interrupted by the speech consonants. They are of a tubal, high-pitched character, *minus any fremitus* conveyed to the ear. In very weak, exhausted patients it is sometimes easier to elicit the whispered voice than deep respirations.

The NORMAL WHISPERED RESONANCE is heard over the lungs as a soft, indistinct, low-pitched, whiffing sound corresponding to the articulations at the larynx. It must be noted, however, that in very many normal chests the whispered voice is not heard over the lung, except quite close to the bronchi. Wherever bronchial breathing occurs pathologically, the extent and degree of the underlying physical changes will be indicated also in the degrees of clearness and intensity of the bronchial whisper.

NORMAL VOCAL RESONANCE.—Considering, now, the spoken voice as heard over the lungs, the ear, directly applied to the chest or aided by the stethoscope, detects an inarticulate buzzing or humming of slight intensity and low pitch, occurring in barely perceptible waves of slightly greater intensity synchronous with the beginning of enunciation of the words uttered.

NORMAL VARIATIONS OF THE VOCAL AND WHISPERED RESONANCE. —Altogether independent of disease, the quality and intensity of the whispered and spoken voice may be modified by perfectly natural conditions. In the very old, for instance, it is louder than in young children, partly because of the louder, graver-pitched tones of the voice and partly, no doubt, because of the "wasting of the pulmonary parenchyma and the thickening and hardening of the bronchi in old age"; and is, at the same time, somewhat of a tremulous or bleating quality. It is lessened in strength in women also, for evident reasons, and in them and in children is distinct only over the upper half of the chest.

The intensity over the whole chest is greater in those who have thin than in those who have fat or muscular, thick walls. It is stronger in front than behind, except in the interscapular region; in the infraclavicular regions than below, and on the right than on the left side. Because of the last-named fact—which is in harmony with the slightly greater vocal fremitus on the right side—the significance of an exaggerated vocal resonance on that side has less definite and decided diagnostic value than a similar change on the left side. Indeed, the same regional variations of intensity hold with respect to the vocal resonance as with the vocal (tactile) fremitus and the breath sounds. Hence the importance of avoiding hastily drawn conclusions from the state of the vocal resonance in any one portion of the chest; on the contrary, only by the comparison of corresponding parts on the two sides, after allowing for natural physical differences, may we expect to infer safely and significantly by this method of examination.

Vocal Resonance and Whispered Voice in Disease.—The modifications met with due to pulmonary affections are essentially those of intensity. The latter element may be slightly increased; it may be moderately or markedly increased (*bronchophony, pectoriloquy*); it may be decidedly increased, with accompanying alterations of quality (*egophony, amphoric voice*); it may be diminished or suppressed.

Both the spoken and whispered voice are similarly affected by the same pathologic conditions, as is the palpable fremitus, and may therefore be considered together. Nevertheless, at the beginning it should be noted that alterations of the whispered voice more often yield delicate and precise results than do those of the loudly spoken.[1] The reason for this is clear when we apprehend the fact that, the articulating voice being produced in the mouth in its passage downward through the respiratory tract to the thoracic walls, it has to "pass through the glottis, where the approximated vibrating cords may be supposed to offer a formidable obstacle to the transmission downward of the spoken words" (Steell). In the case of the whispered voice, therefore, which consists merely of an interrupted (the words), exaggerated expiratory murmur, there is no glottic closure or vibration to interfere with its downward transmission and ready conduction to the chest wall through intensifying areas of disease.

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[1] In the chronic bronchitis associated with senile emphysema and fibroid phthisis, I have found the whisper to elicit viscid mucous râles that were inaudible by the usual respiratory efforts and tests.

(1) Exaggerated Whisper or Vocal Resonance.—This differs from the normal only in a slight increase of intensity. It occurs over *small* or *partial consolidations* of the lung, and is thus a useful sign chiefly in the diagnosis of *tuberculosis*, associated with broncho-vesicular breathing. In addition to the simple exaggeration of loudness, the voice may sometimes seem to come from a part of the lung near to the auscultator's ear. In such cases there may be an atypical unchanged, or even diminished, vocal fremitus. This may be noted in certain cases of pleuritis with moderate, superficial consolidation of the adjacent lung underneath. The whispered voice is especially delicate in the discovery of small consolidations, without definite bronchial respiration or increase of the tactile fremitus.

(2) Bronchial Whisper and Bronchophony.—Whenever the normal or a simple exaggerated whisper or vocal resonance is augmented to a degree that is heard normally over the trachea and primary bronchi, it indicates, over the lung, that *consolidation* of the vesicles has occurred to a considerable extent, usually as in *pneumonia*. Associated with it is bronchial respiration and increased vocal fremitus on palpation. Bronchophony may be elicited also over large, complete *tuberculous condensations* of the lung, over *retracted and compressed lung* above pleural effusions, over *cavities surrounded by solidified tissue*, and *bronchial dilations with thickened walls*, and sometimes in marked *senile emphysema*. The absence of increased whispered or spoken voice in undoubted cases of lobar pneumonitis in the second stage indicates either that the bronchus leading to the consolidated area is occluded by exudate, or that a large pleuritic exudate (pleuropneumonia) intervenes to nullify the increased conductivity of the consolidated area.

The bronchophonous voice and whisper are respectively and relatively not only louder and more concentrated, but manifest a vibrating or wavy quality approaching the actual articulation of the syllables or words used instead of the indistinct, monotonous humming of the normal vesicular sounds.

(3) PECTORILOQUY; WHISPER PECTORILOQUY.—As the double-derived term suggests—chest speech—we have here a degree of whisper or voice intensity, with *articulate* words audible, as if the patient were speaking with his lips directly beneath the listener's ear. It is a pathologic laryngophony, or an exaggerated bronchophony. The whispered pectoriloquy is even more striking or startling than the spoken. The words should be uttered slowly. In a large majority of cases the presence of this sign points to a fair- or large-sized *cavity of the lung*, especially one in free communication with a bronchus and in close approximation to the chest wall. The area of pectoriloquy is sharply defined, as a rule. The loudly transmitted words have a hollow, cavernous quality. However, pectoriloquy may be significant also of *large, dense consolidations surrounding bronchi*, and thus acting as unusually good conductors of sound to the chest wall. Here the pitch of the whispered or spoken voice is higher than over the pulmonary excavations. Other less frequent causes of pectoriloquy are *collapsed lung from pleuritic effusion*, a tumor between the lung and the thoracic wall, pressing upon a bronchus, and an open, *circumscribed pneumothorax*, communicating freely and directly with a large bronchus.

(4) EGOPHONY.—A sign of vocal resonance, to which Laennec attached special importance in his "Traité de l'Auscultation Mediate." It is at once an intensification—a little less than bronchophonous loudness—and a peculiar change in the quality of the voice transmission. This has a distant, nasal, tremulous, bleating character—goat voice. The locality of the sign is limited usually to the region at the level of the angle of the scapula. It is most frequently heard in connection with *moderate-sized pleuritic effusions*, although not invariably so, since, as originally held by Laennec, partial adhesion of the lung to the chest wall may be associated, and in some cases there may be solidified lung beneath a thin layer of fluid. The egophony is best heard just below the upper border of percussion dulness, and often conveys the impression that the fundamental tones of the voice are intercepted, the shriller, high-pitched overtones remaining near the surface as a sort of quivering echo of the others. The sign is not at all a common one of moderate pleuritic effusions, nor is it necessarily always indicative of them, as it is claimed to have been heard in connection with consolidations of the lung without accompanying effusion. The nasal twang timbre of egophony has been attributed

to the partial flattening of the bronchi from compression by the fluid. This view is favored on account of the fact that the sign is absent in small effusions (no compression), and lasts seldom more than from five to seven days because of such an increase in the amount of liquid as to obliterate the lumina of the tubes.

(5) AMPHORIC VOICE.—This is the vocal analog of the amphoric breath sound and amphoric resonance heard over *large, tense-walled, communicating cavities* in the lung or open pneumothorax. The voice has a ringing, metallic or musical tone, of variable intensity and pitch, superadded to the usual hollow character, which may be simulated by speaking into an empty pitcher or jar. In some cases the words, instead of being loud and blurred, may be as articulate as in pectoriloquy.

(6) DIMINISHED OR ABSENT VOCAL RESONANCE.—The same pathologic physical conditions which cause the palpable vocal fremitus to be diminished or checked likewise so affect the audible vocal resonance. Thus, we find the latter of weakened intensity in bronchitis whenever there is much secretion. It is enfeebled, also, in *emphysema*; in acute plastic or chronic *pleuritis with thickening of the membranes*; sometimes in *fibroid phthisis with thickened pleura*; and, when occurring persistently all over one lung, other causes being excluded, may signify obstruction of the supplying bronchus by an *aneurismal or malignant tumor.* Finally, it may be noted during the progress of dilating pleural conditions which sooner or later totally suppress the voice transmission.

Absence of vocal resonance is met with in total *occlusion of bronchi*, as in the "massive pneumonia"; also in cases of *pleural effusion* of large amount, whether of air (pneumothorax), pus (pyothorax), serum (hydrothorax), or blood (hemothorax): they all act as insulators, interfering with the conduction of voice vibrations to the chest surface. Very thick, old, dense pleuræ may likewise annul the vocal transmission.

Baccelli's Sign.—This is the use of the whispered voice, to differentiate a serous from a purulent pleural effusion. The conditions are not always favorable, nor is the observation, therefore, conclusive or reliable in all cases that the transmitted whispered voice is characteristic of serous, and the non-transmitted voice of purulent, effusion (empyema). For example, the whispered voice may be totally suppressed in very large serous exudations; and, on the other hand, in certain cases of small, recent collections of pus in the pleural sac, the voice conduction is not completely abolished.

Artificial Vocal Resonance; Plegaphonia.—A method devised by Scherwald whenever, for diagnostic and therapeutic reasons, it is imperative to simulate the voice sounds. This emergency may arise in persons who are constantly dumb (aphonic), or unconscious, or exhausted, or whom, as in pulmonary hemorrhage (hemoptysis), it is inadvisable to have speak. The procedure consists in practising hammer-pleximeter percussion over the thyroid cartilage of the larynx or the upper trachea. The tapping should be gentle, with the patient's mouth closed. While this is being done by an assistant, we auscultate the artificial vocal vibrations, as it were, thus set up, preferably during expiration. The sounds produced by these blows obey the same laws of transmission, exaggeration, lessening, and suppression, according to the physical conditions and changes within the thorax, as with the capable, conscious, adaptable phonation.

(D) *Auscultation of the Cough: Tussive Resonance*

Cough, in itself, is an objective symptom, but becomes a physical sign when auscultated in relation to the phenomena which it develops or modifies within the chest. It has much less value and reliability, however, than the vocal resonance. It is less convenient and more difficult to observe the effects of cough as a simple matter of technic, owing to the agitation and movements of the patient's body. The latter should be held as firmly as necessary during immediate auscultation. The chest piece of the stethoscope should also be watched carefully during the act, so as to avoid friction and other extraneous sounds.

The character and intensity of the cough resonance varies, *ceteris paribus,* according to the same physical conditions and laws that the vocal resonance does.

In a *healthy* individual, while in the act of forced coughing, the laryngeal or tracheal sound, as heard with the stethoscope, is rather hollow, and of a pitch and intensity varying with the person's vocal gravity and strength. Over the pulmonary regions the act is attended with a quick, short, dull, indistinct, and diffused sound, without hollow or tubular character, or accompanied by any sensation of fremitus or succussion within the thorax. The external, sudden, expiratory jarring movement of the chest is, of course, quite obvious.

In *disease,* the modifications of the tussive resonance are termed—now familiarly—*dry* or *moist, bronchial, cavernous, amphoric.*

The resonance of the dry cough is harsh, hacking, or brassy, or like a distant "barking." The moist sound is greatly modified by the character and location of the admixed râles. With the stethoscope, a hissing sound is heard to accompany nearly every cough.

The harsh, concentrated bronchial cough resonance of consolidation; the hollow, "sepulchral," sometimes gurgling of certain pulmonary cavities, and the loud, metallic, reverberating resonance over certain other cavity conditions, all associated with sensation of thrill or fremitus from the interior, may readily be understood as mentioned. The irritative cough of pleurisy with moderate effusion may also have an egophonic resonance.

Mere mention may be made again here of the post-tussive suction, or "india-rubber-ball" sound heard with the first inspiration after a cough, suggestive of cavity with soft, yielding walls, especially when accompanied with mucous clicks and moist râles.

In conclusion, reiteration of the service of cough as an adjuvant to auscultation may be made. That is, to *summarize*, after repeated coughing inspiration is deeper and the respiratory murmur louder; mucous obstruction of the bronchi is removed, so that previously indeterminate breathing becomes more distinct and definite, vesicular or bronchial, as the case may be; râles are elicited, intensified, shifted, numerically increased or diminished, qualified.

EXPLORATORY PUNCTURE OF THE PLEURA (THORACENTESIS)

Supplementary, and yet not infrequently necessary and valuable in the direct physical diagnosis of pleural effusion, and its differential diagnosis from other conditions, is the procedure commonly known as *exploratory puncture* of the pleura. Under strict aseptic precautions, the attached needle of a hypodermic syringe, or the small-calibered trocar and canula of an aspirator, is plunged vertically into an interspace—usually the seventh, in the midaxillary line—close to the upper border of the lower bounding rib, so as to avoid the intercostal vessels. If the point be immersed in fluid, the withdrawal of the piston or aspirator will be followed by an immediate rush of the effusion into the barrel of the syringe or into the aspirator bottle. The negative value of a "dry tap" depends upon the length of the hypodermic needle, if that be used, as in certain cases of old, greatly thickened pleura its point may not penetrate to the liquid within. Again, the exudate may be so thick and flaky as to close the exploratory needle. Of course, in acute or chronic pneumonia, merely thickened pleura, or tumors of the chest, the syringe draws nothing, perhaps, but a drop of blood.

When the *differential diagnosis* can be made unmistakably by the usual methods of physical exploration, it may nevertheless be important to practise thoracentesis, in order *to determine the nature of the liquid*, by the microscopic, chemic, and tinctorial and cultural methods described in works on clinical or laboratory diagnosis. Thus the histologic, chemic, and bacteriologic constituents are ascertained.

COMBINATION OF THE PHYSICAL SIGNS: THEIR ASSOCIATED SIGNIFICANCE

It must be evident to the student of the preceding pages that any comprehensive and accurate diagnosis of the physical conditions and changes of diseased lungs cannot depend upon the results of but one method of examination, still less upon any single, isolated physical sign. Rather, one must, as it were, "strike a balance" of preponderating evidence favorable to a physical diagnosis, based upon a careful comparison of *all of the signs* perceived by means of *all of the methods* used. This embraces, in other words, the searching analysis of the relative value and significance of the combined physical signs, and of the methods by which they are derived, respectively, in connection therewith; and, further, a rational, judicious synthesis of these separate deductions into a positive or probable and harmonious whole; this conclusion is the diagnosis.

This intellectual process has to deal, it must be remembered also, with three sorts of physical signs in practically every case examined, bearing upon the statement of the preceding sentence. These are: *first*, the absolutely clear, definite, unimpeachable ones; *secondly*, those of doubtful, indefinite, more or less probable manifestation—usually the signs of developing and changing pathologic states; *thirdly*, the absent signs—those which have certain value as negative evidence when the complete picture for a diagnosis is wanting in the apparent and suggestive signs.

An understanding of what has gone before may be facilitated by referring to the following table, slightly modified after Da Costa:

Association of Physical Signs

PALPATION.	PERCUSSION.	AUSCULTATION.		PHYSICAL CONDITION.
Fremitus.		*Respiration.*	*Voice.*	
Unimpaired.	Resonant.	Vesicular murmur or its modification.	Normal vocal resonance.	Lung tissue healthy or nearly so; at any rate, no increased density from deposits or from pressure.
Increased.	Dull.	Bronchovesicular or bronchial.	Bronchophony.	Consolidation of lung structure.
Diminished or absent.	Dull.	Absent or very distant.	Diminished or absent.	Pleural effusion.
Uncertain; mostly diminished.	Vesiculo-tympanitic or tympanitic.	Feeble or cavernous, according to cause.	Uncertain; diminished or cavernous.	Increased quantity of air in lung or lungs due to over-distention of vesicles, or to a cavity.
May be diminished.	Amphoric or metallic.	Amphoric or metallic.	Amphoric or metallic.	Large cavity with tense, elastic walls.
Uncertain.	Cracked-pot sound.	Cavernous.	Cavernous.	Cavity communicating with a bronchial tube.

From the above it will be observed that the signs of cavity in the lung are characteristic in that the cavernous or amphoric quality of sound attends uniformly the percussion and auscultation; and it may be said, in addition, that amphoric phenomena accompany the three acts of breathing, of coughing, and of speaking. This is true also of the adventitious sound known as metallic tinkling.

The combinations of physical signs given in the table are those usually met with in adults; in children they are less evident and constant, because of the thin, elastic chest walls, principally, the very clear lung sound making it especially difficult to elicit dulness on percussion. Again, the small size of the chest, and the fact that most of the pulmonary diseases causing any dulness are bilateral, make comparison of the two sides less valuable both by palpation and percussion. Auscultation is, therefore, the method most suitable, and to be applied first, before the child is apt to cry. The crying voice may then be listened to as the vocal resonance.

This variability and uncertainty of the physical signs is a marked feature of the condition known as pulmonary collapse or atelectasis, where the small areas of relative dulness at the bases of the lungs posteriorly, simulating bronchopneumonia, may within a few hours give way to clearness, to be replaced in the same regions, or elsewhere, by impaired resonance again within a short time. This is explained by the fact that in cases of capillary bronchitis, temporary exudative obstruction frequently causes collapse of the lobule, the supply of air being cut off and the residual air being soon exhausted. Subsequently the expulsive power of a violent expiration may drive out the mucous plug, as during a paroxysm of coughing, and the naturally clear, resonant note is heard once more. It is usual to find associated with atelectatic dulness absent respiration or faint, distant bronchovesicular breathing, differing thus from pneumonia with its superficial, harsh, bronchial breathing. On inspection, also, labored inspiratory efforts and recession of the lower interspaces are witnessed, with collapse of lung often coming on suddenly.

Finally, it must be admitted that the conditions in certain cases of pleuritic effusion are such that a pneumonic consolidation may be simulated. In the absence of thickening of the pleura, and with an amount of fluid sufficient to cause considerable compression of the lung, the transmission of bronchial breath sound and bronchophony may be quite suggestive of pneumonia. If, however, the pleura is thickened by a simultaneous fibrinous deposit; if the liquid is dense, as may be determined by exploratory puncture; and if other organs, as the heart and liver, show displacement, pleural effusion is present, whether or not pneumonia is responsible for the bronchial breath and voice sounds. The absence of râles, also, at least of consonating râles, confirms the pleuritic causation.

SECTION II

SPECIAL PHYSICAL DIAGNOSIS OF SOME DISEASES OF THE RESPIRATORY TRACT, INDUCTIVELY CONSIDERED

CHAPTER VIII

HYPOTHETICAL AND RECORDED CASES

THE student having acquired an elemental and general knowledge of the facts and principles concerned in the physical diagnosis of pulmonary and pleural affections, with their illustrative applications, he is now presumably ready to take up a study of the method of arriving at a complete diagnosis of the special diseases with all the findings before him. It is here deemed more natural and rational and cultural to approach such study—assuming the general familiarity just referred to—in the manner that each one must necessarily and responsibly do sooner or later, at any rate; that is, depending upon his observational and reasoning powers, learn all he can, and think and decide as best he can about the physical signs of each case as he meets with it in the hospital dispensary or ward, the bedside in the home, or within his own consulting office.

It is desirable to begin the habit, as early as possible, of inducing (literally, *leading into*) a diagnosis by an analysis of the collected, clearly observed physical signs, as one must really do in practise, instead of merely memorizing lists of physical signs under the headings of the special diseases in which they occur respectively, endeavoring later to make the case fit one of the lists by inclusion or exclusion—a fallacy and too common procedure to be unlearned that were better anticipated and prevented.

Case No. 1.—A young adult, male, weaver by occupation, working in a dusty atmosphere, calls at his physician's office two days after having been exposed for several hours in the mill to a draft of cold, moist air, in March. He complains principally of a sense of weight, tightness, or soreness behind the sternum, a tickling in the throat or below the suprasternal notch, followed by an irritating, dry, harsh cough, often paroxysmal, and causing pain in the muscles of the chest, especially along the costal margins; the cough is worse at night, on lying down, and again in the morning, on rising; some oppression of breathing; all of which points to an acute or subacute inflammation of the upper respiratory tract.

PHYSICAL EXAMINATION.—*Inspection, palpation,* and *percussion* are practically negative, although at times a slight increase in the frequency and depth of the respiratory movements is visible. On *auscultation*, the vesicular respiratory murmur is everywhere normal; over both infraclavicular regions, however, near the sternum, the bronchial respiratory element is a trifle harsher than normal, but fails to obscure a few low-pitched, groaning or sonorous râles, and occasionally a sibilant râle a little higher up and farther out on the right side, these râles being inconstant, appearing and disappearing with cough, deep breathing, and whispering. The *vocal resonance* is normal.

ANALYSIS OF THE PHYSICAL SIGNS.—In the first place, such a walking case, minus the evidences of severe illness, in spite of the acuteness of the attack and the subjective localization of the discomfort and pain to the region of the main bronchi, precludes the probability of any *pulmonary* involvement of acute character. This view is quite confirmed by the absence of any visible (one- or two-sided), palpable, or percutory signs of physical changes in the lungs—no alterations of the size, shape, symmetry, or respiratory movements of the chest suggestive thereof, nor, indeed, any of the vesicular breathing or voice sounds; simply a roughness, bilateral, of the bronchial breathing where normally this type is heard, indicating an irritative, congestive swelling of the mucous membrane, and *râles*, due to the formation of a viscid secretion in the bronchi, heard during inspiration and expiration, with the other characteristics of purely bronchial origin, the solitary sibilant râle indicating extension of the catarrhal inflammation to a branching tube of small size extending into the right apex.

A few days later the patient reports feeling much less substernal soreness and oppression, and the expectoration of a thick, yellowish-white, slightly frothy sputum, and reexamination elicits but one change in the physical signs—the dry râles are replaced by bubbling sounds, of similar behavior.

Diagnosis.—We have determined a twofold pathologic fact, namely: (*a*) inflammation of the bronchial mucous membrane; (*b*) its limitation (with the single exception on the right side) to the main bronchi, probably including their bifurcation: a *simple bronchitis.*

Case No. 2.—A man, aged forty-four, merchant, during the prevalence of cold, moist, penetrating March weather, is taken suddenly ill at midnight, with a sharp chill, general aching and depression, stabbing pain ("stitch") on the right side, dry, short, restrained cough, rapid respiration and pulse, and fever. The pain is located in the axilla, and as far forward as the nipple; is aggravated both by breathing and coughing. The patient is seen by the physician for the first time nine hours afterward.

Physical Examination.—On *inspection*, the patient is discovered lying on the right (affected) side; a circumscribed, almost livid flush on each cheek, a trifle deeper in shade on the right; the breathings extremely frequent, panting in character, with occasional expiratory grunts. There are no abnormalities of contour of the chest. The respiratory movements, however, are broken, and restricted somewhat on the right side, especially at the lower half, and during the inspiratory act. This is perceived to affect the expansion as well as the elevation of the chest. The movements on the left side are clearly exaggerated, and slightly so in the right infraclavicular region.

Palpation shows the vocal fremitus to be comparatively normal, although, if anything, but very slightly diminished over the right lower as compared with the upper zone.

Percussion on both sides reveals a clear note, differing in the particular quality on the two sides, however. On the right side it has a higher-pitched, empty, vesiculotympanitic, almost cracked-pot tone; on the left, a lower-pitched, rather hyperresonant tone.

Auscultation exhibits a weakened respiratory murmur in the infrascapular and axillary regions on the right side; over the upper lobe it is a little harsher than normal; all over the left lung the quality is vesicular, but intensified, and at the same time notably continuous, while on the opposite side (the one complained of) it is interrupted and irregular in rhythm. Quite distinctly heard over the right base, in addition, are some fine, dry, crepitant râles at the end of inspiration. The vocal resonance is everywhere normal.

Fourteen hours later, at 11 P.M., the distress, pain and dyspnea, and other symptoms being aggravated, no change in the physical signs is observed except a greater abundance and prominence of the crepitant râles, with, over the right lung base also, the feeble vesicular murmur now replaced by a somewhat harsh, prolonged, bronchovesicular respiratory murmur.

The next (second) day, at noon, with harassing subjective and objective symptoms, the cheek flushes deeper and duskier, orthopnea pronounced, and the distressing cough accompanied with a tenacious, sort of brick-red or rusty sputum, the physical signs are found to be as follows:

Inspection notes a marked inequality in the movements of the two sides; that on the right is more restricted than at first observed, the lagging and relative inaction giving it the appearance of fulness and weight, while on the left side the excursion of movement is, by contrast, greatly increased. But the movement of elevation is not so much diminished as that of expansion. The abdominal movement on the right side is likewise visibly obstructed.

Palpation now elicits a positive increased fremitus over the whole right lower lobe, posterolaterally, both as compared with the upper portions and the opposite side. Touch confirms, also, the signs detected ocularly.

On *percussion*, over the right side anteriorly a vesiculotympanitic note persists (Skodaic resonance); posteriorly, below the scapular angle and extending into the lower axillary region, the sound is dull, passing into the liver dulness to the borders of the ribs. On the left side, front and back, the note is quite hyperresonant; thus is manifested three degrees of pitch and loudness, as well as quality, namely, low-pitched, loud sound on the left side, medium-pitched, moderately loud (semitympanitic) sound over the upper part of the right chest, and high-pitched, least loud sound at the right base. In the latter region the sensation of resistance is increased.

Auscultation of the region of dulness shows a superficial, loud, harsh, tubular breath sound, the expiratory sound as high in pitch and as long in duration as the inspiratory (bronchial breathing). Above, on the same side, the breathing is simply exaggerated vesicular, although, as we approach the level of the seventh rib downward, the sound becomes bronchovesicular. On the left side throughout, marked intensification of the vesicular murmur is heard. At the right base we find a distinct increase of the loudness of the vocal and whispered resonance (bronchophony).

SYNTHETIC ANALYSIS.—The acute character and onset, and location of the symptoms point to an undoubted severe pleural or pulmonary inflammation, or both, on the right side.

This inference is further confirmed by the disturbed respiratory movements observed on the right side, indicating at least a painful, if not a mechanical, interference with breathing, rather pleural in origin with the predominance of the former condition; of simultaneously combined pulmonary origin with growing predominance of dyspnea, from exudative obstruction to the entrance of air into the vesicles.

The percutory and auscultatory signs (first examination) indicate a relaxed state of the right lung (tympany) and compensatory activity of the left (hyperresonance: exaggerated respiration). That this relaxation of lung is immediate is proven by the fact that if not so the location of its determining signs would be higher (mediate relaxation from pleural effusion below, with visible and palpable evidences of this below), whereas the respiratory changes and crepitant râles heard at the base can mean alone involvement of the lower lobe, with some accompanying pleuritis sicca, no doubt. Combined, these signs point to the one acute cause of such relaxation of lung, namely, *congestion.*

This inference is corroborated at the second visit to the patient, with the additional signs of beginning exudation into the alveoli, and even closure of some lobules already.

Upon scrutinizing the results of the third physical examination, the only deduction must be that of a rapid formation of complete lobar consolidation, essentially because of the localized exaggerated vocal fremitus and resonance, bronchial breathing, and dulness, according to the established acoustic law of the better conduction of soundwaves in full amplitude through solid than through air-containing structures.

DIAGNOSIS.—*Acute croupous* or *lobar pneumonitis*: The *first stage,* or *stage* of *congestion,* and the *second stage,* or *stage* of *red hepatization.*

With the patient gravely ill, these physical signs persist for seven days. A sudden crisis in the symptoms occurs; the temperature drops to subnormal, general prostration is intense, and cardiac collapse and a fatal issue are imminent. The natural powers of the system, however, aided by appropriate and prompt treatment, enable the patient to withstand and survive the shock and depression. And still there is no change in the physical signs—nor in one's anxiety, perhaps.

On the second day after the critical condition just mentioned, the patient breathing much easier, the sputum grayish or yellowish, less thick and rusty, and more abundant and frothy, reexamination shows less marked exaggeration of respiratory movement on the left side, no jerkiness on the right side; diminished prominence of the vocal fremitus over the right base; dulness remains; the bronchial breathing is not so sharp, but more distant, though quite characteristic; new râles have appeared, however, of a moderately fine, moist type, heard both with inspiration and expiration.

At the end of two weeks from the time of the third examination recorded, very slight restriction of the right-sided thoracic movement is noticeable, and no exaggeration of movement on the left side at all; vocal fremitus is practically uniform everywhere; a relative dulness over the affected lobe is now noted, with the peculiarity that the partly clear element is tympanitic in quality; hyperresonance over the right upper lobe and over the left lung has disappeared; bronchovesicular breathing at the right base, here and there ill-defined, and coarser moist râles, mingled with some fine crepitations, are also audible.

All of which points unmistakably to the transition of the case from the *third stage*, or *stage* of *gray hepatization*, to that of *resolution.*

That clinical recovery and pathological recovery are not synonymous, nevertheless, is seen in the fact that two months later, the patient having been well and able to work as usual for nearly five weeks, the following physical signs remain: in the right infrascapular region only, slight impairment of resonance, with sensation of resistance also slightly above that of the corresponding left-sided space, and, besides, a tympanitic character of clearness, while on auscultation the breath sounds are a little less clear, near, and loud, yet, withal, the expiratory portion is somewhat prolonged and low-pitched.

This condition of affairs is readily explicable as due to, in the first place, slight pleural thickening which accompanies practically all cases of pneumonia, from extension of the inflammation to the surface and a lobar pleuritis resulting which leaves the pleura a trifle less thin, smooth, and translucent than normally; hence the percutory signs. The fact that the impaired clearness of note is pleural instead of pulmonary is evident, also, in the auscultatory indication that air is free to enter the vesicles over the same location. But that these vesicles have not regained their normal resiliency is equally apparent in the persistence of a tympanitic element and a prolonged expiratory sound on percussion and auscultation, respectively—significant of relaxed lung tissue.

VARIATIONS.—Before considering the differential diagnosis of lobar pneumonitis, certain variations of the physical signs not met with in the average, typical case just narrated should be pointed out, because they enter more or less largely into this question, and their recognition and estimation constitute important items of resource in differentiating as accurately and readily as possible.

Age is a factor. When pneumonia attacks a debilitated old man, most of the physical signs are retarded and insidious in their appearance and development, and lack distinctness. This is particularly evident as regards the fremitus and dulness, and, though less marked, the occurrence of bronchial breathing, which may be distant and rather indeterminate. In very young children, also, a considerable solidification may exist without dulness; indeed, there may be tympany instead.

Sex and Build.—Increased tactile fremitus, which is an important factor in the diagnosis, may be absent on account of the weaker voice in women, or because of a thick, fat chest wall.

The *location* and *extent* of the consolidation may influence the physical signs. Thus, in the more infrequent cases of *apical* pneumonia, well-marked dulness is rarely met with. Bronchial breathing is likewise less obvious. In the so-called "*central pneumonia*"—which, by the way, is the variety most often met with in the aged—the deep-seated area of hepatization may escape detection so far as percussion is concerned; and even on auscultation, the bronchial breathing may be so feeble and distant as to be recognized only with great difficulty and care. The pneumonitic process may reach the surface later, however, and then be more easily diagnosticated. Small areas may so be discovered, beginning high up in the armpit; in doubtful cases, therefore, this part of the axillæ should always be examined with closeness. Pneumonia at the left base sometimes escapes early and definite detection on account of the proximity of an enlarged stomach, giving rise to a tympanitic note.

Massive pneumonia—the term applied to that variety in which the bronchus leading to the consolidated lobe or lobes is occluded by exudate—may be particularly puzzling because of the absent fremitus and bronchial breathing, and the more than usually dull and resistant sound, as in cases of pleural effusion.

"*Wandering*" or "*creeping pneumonia*" (also *migratory*), as the term signifies, is a form of the disease in which the signs of consolidation are found to extend from one lobe to another, or to disappear over one lobe and appear over another. This is more properly a pneumonia with relapses than an actual wandering of the inflammation.

Again, it should be remembered that quite frequently cases of pneumonia are first seen when the first stage (congestive) has passed; that *egophony* may sometimes be heard over the consolidated lobe; that both dulness and bronchial breathing may be transmitted a short distance beyond the actual borders of consolidation, especially when, as in the majority of cases the right lower lobe is affected, the enlarged as well as solidified lobe is in close contact with the spinal column, so that a narrow zone of moderate bronchial breathing may be audible along the column on the *sound* side; that the disease may run a short, even two-day course (*abortive form*); that it may also manifest a prolonged persistence of the physical signs, from (*a*) delayed resolution, (*b*) relapse, or a modification of them (*c*) because of a terminal transition to abscess or gangrene of the lung.

Finally, while as a complication of other diseases regular pneumonia may be seemingly present symptomatically, the physical signs are so irregular as to render the diagnosis between the lobar and catarrhal varieties uncertain in some cases. This is especially apt to occur in connection with epidemic influenza. A patch of limited or incomplete consolidation may be found between the angle of the scapula and the spine, or in the upper axillary regions, as indicated by the dulness and bronchial breathing. More often, however, the physical signs of the so-called *influenzal pneumonia* are those of an intense acute *pulmonary congestion*, and true hepatization does not occur. There may be no perceptible alteration of the percussion note, certainly no dulness, and usually nothing more than a general slight impairment of resonance over one lung. Neither is bronchial respiration evident; on the contrary, only a few areas of bronchial harshness (irritative) in the midst of a general area of moderate suppression of the breath sounds. Crepitant or some fine moist râles may be heard.

The pneumonias which occasionally follow surgical and accidental injuries and etherization are of this character.

Reference has been made to the fact that every pneumonia in which the inflammatory process extends to the pleural surface of the lobe is associated with a certain fibrinous pleuritis. But in some cases this latter condition may become so severe and extensive (*pleuropneumonia*) as to give rise to physical signs so predominant that those of the pneumonia may be obscured almost beyond recognition. This will be pointed out more fully below in differentiating pneumonia from pleurisy with effusion.

DIFFERENTIAL DIAGNOSIS.—In the first stage of pneumonia the signs may be simulated by three pathologic conditions, namely, *acute*

pulmonary congestion (see under the preceding heading), ***hypostatic congestion***, and *pulmonary edema.*

(1) *Acute Pulmonary Congestion.*—In the first-named, the bilateral, moderate harshness of the respiratory murmur soons ends as it began, while in pneumonia the localization of the congestion to the lobe that is to be consolidated is manifested by the transition within twenty-four or thirty-six hours of a rather weakened localized murmur to true bronchial breathing, maintained until the crisis, unlike the general congestion, the mentioned signs of which may be intermittent. In the simulating affection, also, the crepitant râle is much less constant, scanty, generalized, and recurrent, whereas in pneumonia it is common, abundant, localized, and recurs only during resolution (*râle redux*), if at all.

(2) *Hypostatic Congestion.*—In the course of long-continued fevers, as typhoid, and of chronic debilitating diseases attended with weak heart, causing the patients to be bedridden in the dorsal recumbent position, such as cancer; paralysis, especially that following cerebral apoplexy, and in the later stages of diabetes, Bright's disease, and tuberculosis, lobar pneumonia may be suspected as a complicating affection when the simulating signs are really due to a stasis or engorgement of the dependent (postero-inferior) parts of the lungs from the combined factors of a weak, flabby right heart and the influence of gravitation. In very protracted cases the congestion may actually be transformed into a *hypostatic pneumonia*, a certain quantity of exudation expelling considerable air from the vesicles and infiltrating some of the interstitial tissue.

Here, apart from the suggestiveness of the underlying disease, usually easily recognized, the physical signs of a hypostasis may be differentiated in the following particulars, principally: (*a*) on *inspection,* while the respiratory movements are somewhat shallow (except in the infraclavicular regions), there is not the painful, one-sided restriction of movement seen in true lobar pneumonia, with exaggeration on the opposite side, and the acute dyspnea is absent; (*b*) the *vocal fremitus* is feeble at the base, normal above anteriorly; (*c*) there is slight dulness over both bases, frequently commingled with a slightly tympanitic quality; (*d*) on *auscultation* we hear the breath sounds, likewise those of the voice, diminished. Fine moist râles may be audible, due to adjacent bronchitis. At times the expiratory murmur is slightly prolonged, and in the terminal cases of hypostasis, where some induration has occurred, the breathing may have a bronchial quality. An important distinguishing point is the fact that hypostatic pneumonia is invariably bilateral, while the ordinary acute pneumonia is rarely so. Not infrequently the evidences of congestion (hypostatic) are a little more extensive on the right side, the relative dulness reaching as high, perhaps, as one-third the way up the scapula.

(3) From *pulmonary edema* acute lobar pneumonia may be differentiated by the conditions under which the former occurs (as a part of the general dropsy of cardiac or renal disease, and in the terminal stages of chronic anemic and cachectic conditions); by the bilateral impaired resonance and weak breath sounds below the scapular angles, and especially by the distinct fine moist râles. As the serous effusion into the alveoli increases and extends to the higher portions of the lungs, more marked dulness and bronchovesicular respiration may be heard, and the liquid râles become less uniform on account of the admixture of coarser (slightly) râles, which may be heard also in the axillary regions.

As pulmonary edema is often preceded in transition by hypostatic congestion, and may thus be a precursor of death because of the greater loss of strength and vitality indicated, for prognostic and therapeutic reasons it is important to recognize, if possible, this transition. As a rule, in edema the dyspnea is more marked, and becomes rapidly aggravated; dulness on percussion is more decidedly evident, and unassociated with a tympanitic quality, and, most distinctive of all, the presence of more or less fine moist râles.

The differential diagnosis of the second or consolidation stage of lobar pneumonia is seldom difficult, the only diseases of importance simulating it being *pleurisy with effusion* (*acute*) and *acute pneumonic phthisis* (tuberculosis); rarely, an *hemorrhagic infarction* of the lung also. The first of these will be considered later.

(4) *Acute Tuberculopneumonic Phthisis.*—It may be impossible, for the first week or ten days, to distinguish this from ordinary croupous pneumonia by physical signs alone. The signs of pneumonia occurring in a small-chested individual should invariably lead to a careful examination of the apices, where often the tuberculous focus causing the acute outbreak of extensive consolidation in the lower lobes may be discovered. In acute tuberculous pneumonia, again, evidences of bilateral involvement are more frequent. Instead of a critical decline about the eighth or tenth day, the disease persists, minus the physical signs of resolution as in the lobar pneumonia; on the other hand, soon plus those of softening and cavity formation, namely, cavernous or amphoric breathing, gurgling râles and metallic

tinkling, and circumscribed areas of percussion tympany. Otherwise, the differentiation must be made by an examination of the sputum, and consideration of the clinical aspects and personal and family history of the patient.

(5) In *hemorrhagic infarction* the signs of consolidation point to a small, circumscribed area, usually in the middle or lower lobe of the right lung, near the surface, or perhaps at one of the borders. A deeply seated, centralized infarct, with the symptoms of lobar pneumonia at the onset, is seldom recognizable by the methods of physical examination. A superficial infarct, however, gives rise to a limited area of dulness on light percussion; the fremitus is, if anything, slightly increased, unless complicated by pleuritis, and some localized crepitant râles and bronchovesicular breathing may be heard. Associated with these signs are usually found those of valvular heart disease, especially when due to ulcerative or malignant endocarditis. Other septic or pyemic diseases, infectious fevers, and the puerperal state and traumatism causing *venous thrombosis* of the leg, uterus, etc., may be present as suggestive of infarction from an embolus rather than of a true pneumonitis.

Case No. 3.—An elderly gentleman, apparently about fifty-eight or sixty years of age, enters one's office, manifesting some shortness and laboriousness of breathing, and embarrassed by brief, harsh, at times slightly rattling, explosive spells of coughing attended with a scanty mucous expectoration. His lips and cheek eminences show some lividity, and over the latter the bluishness is marked by a network of dilated capillaries. He stands and walks slightly bowed, and is rather bulky-chested, although not large-limbed. When in his teens he suffered from frequent attacks of asthma for several years, and ever since twenty-four or twenty-five years of age, until twenty years ago, worked extremely hard physically (in iron works). He has also been subject to more or less severe attacks of bronchitis, lasting from one to two months, nearly every winter. He comes for the relief and cure, if possible, of the dyspnea and cough, especially, and the mitigation of his condition and of the recurrent bronchitic attacks generally.

Physical Examination.—*Inspection* of the bared chest shows the following: The peculiar breathing movements of the thorax attract one's attention at once, but, confining our interest to the *size* and *shape* of the thorax first, we note that it is generally enlarged and rounded, the enlargement being most marked in the upper half. The whole chest is higher than normal, too, and the neck appears correspondingly shortened and thick. The stooping posture is augmented by the elevated and rounded shoulders, and the evident increase in the anteroposterior diameter, as well as by the exaggerated curve of the dorsal spine. The point of greatest curvature corresponds to the lower portion of the thoracic spine. The sternum is conspicuously prominent, arching forward a little more noticeably at level of insertion of the fourth rib. The bone appears unusually broad, however, and flat at the angle of Louis—i. e., the angle formed by the planes of the manubrium and gladiolus is more obtuse.

The sternomastoids and scaleni antici stand out prominently, and the jugular veins (external) likewise, from distention. Evanescent tumors are seen rising and falling above the clavicles with violent coughing attacks; at other times the supraclavicular regions are depressed. The scapulæ are widely separated. The intercostal spaces are widened, especially at the upper part of the chest. While the epigastric angle is also widened—obtuse—the lower portion of the chest seems transversely constricted (*peripneumonic furrow*) because of the comparatively greater increase of all of the upper diameters. The heavy ribs are not uniformly arched, but present conspicuous angulations at various points; they are also rigidly and horizontally arched forward, as in a fixed inspiratory position, especially above the sixth rib.

The general respiratory expansion is distinctly diminished; there is absence of resiliency of movement, the ribs and sternum moving upward (principally) and forward (slightly) as if made of one piece. With the elevation of the chest the hypertrophied sternomastoids and scaleni jerk out tensely into greater prominence, the supraclavicular, suprasternal, and upper intercostal spaces being correspondingly deepened with the inspiration. The expiratory movement is, on the other hand, tardy, slow, and labored, and the spaces just mentioned bulge out during forced expiration. The lower part of the chest, including the diaphragmatic region, is seen to be retracted during inspiration, and the upper bellies of the recti muscles firmly contracted; indeed, the breathing movements altogether are chiefly abdominal and reversed as regards the excursions of the mural tissues and the acts of respiration. The diaphragmatic shadow (Litten's sign) is faintly seen, moves but an interspace, and begins at the eighth interspace. During quiet breathing the sign is absent.

Mensuration, with the tape line around the chest at the nipple level, demonstrates *diminished respiratory expansion*: a difference of but 1½ in. between the measurements of deepest inspiration and deepest expiration. The total circumferential measurement of the thorax is disproportionately large: the patient's stature measures 5 ft. 8 in., the thoracic circumference, 41 in., and the man is not obese or otherwise unusually large in build.

On *palpation*, the signs obtained by inspection are confirmed; the extreme hardness and rigidity of the whole bony and cartilaginous thorax, the lack of resiliency and expansibility, are especially noticeable. The vocal fremitus is bilaterally diminished. Some rhonchal fremitus is felt over the region of the main bronchi in front immediately after a coughing spell. Incidentally, the cardiac apex-beat is very feebly palpable, while a moderately marked pulsation in the epigastrium is noted.

Percussion shows a distinct hyperresonance over both lungs, with a boardy or woodeny quality. Near the sternum the sound has a slightly tympanitic tone. The quality of the sound is not changed either by a forced inspiration or expiration. This clear, loud, low-pitched resonance is discovered to extend beyond the normal boundaries of the lungs; with slightly higher pitch it is heard as low as the seventh rib in the midclavicular lines, the ninth interspace in the midaxillary, and the twelfth rib in the scapular lines. With the exception of a small, indistinct area of dulness below the fifth rib, between the sternum and the left mammillary line, no cardiac dulness can be found. Over the sternum itself the percussion note has a marked "box tone" (*Schachtelton*). The splenic dulness is very small and indistinct, and begins at the tenth rib. The apices manifest a vesiculotympany on percussion that is best perceived during the bulging of inspiration. The mobility of the lower borders of the lungs, as revealed by percussion at the end of the two forced respiratory acts, is practically nil.

Auscultation.—The breath sounds over the vesicular lung substance are everywhere soft, and so feeble in places as to be scarcely audible. The most striking feature is marked relative prolongation and weakness of the expiratory sound, with lowered pitch, the shortened inspiratory sound being apparently only about one-third as long as the expiratory. A few sonorous and sibilant râles are heard near the region of the left bronchus, and occasionally some high-pitched wheezing during expiration near the vertebral borders of the scapulæ, as well as a few crackling and fine moist râles near the bases of the lungs posteriorly. The adjacent heart sounds are much enfeebled at the normal position of the apex. The second sound at the pulmonary valve area, however, is relatively sharpened in tone. There is no distinct change in the vocal resonance, although if anything it is a trifle diminished.

SYNTHETIC ANALYSIS.—Inspection and mensuration show obviously enough, without any lengthy discussion, the presence of some intrathoracic condition of chronic or permanent structural change as the cause of so great and general enlargement, and yet so little power of expansion, even with the manifest laboriousness of effort—a distention of capacity with restriction of activity. Sometimes we meet with large chests in those who have been athletes or devotees of "physical culture," but the normal contour and proportions are maintained, and the respiratory expansion is increased instead of diminished. Here the almost fixed enlargement means that the pleural sacs are distended with too much intrapulmonary air, or with a bilateral effusion of liquid or air. The last-named condition practically never occurs; a double hydrothorax is very rare, and so here presumptively improbable; besides, a pneumothorax is usually unilateral and associated with the signs of advanced pulmonary tuberculosis, and the bulging of a pleural effusion is most marked, and likely to be limited to the base of the chest.

In addition to the detailed physical signs, visible and mensurable, that correspond closely with the characteristics of the emphysematous chest previously described, the inference of an air-containing, large-lunged condition is confirmed by the extensive area of loud vesiculo-tympany on percussion. But we know also that such a protracted history of pulmonary strain as this patient gives, with his having had asthma and chronic bronchitis (râles) for so many years, must have left its impress permanently in weakened elasticity as well as distended capacity of the alveolar walls, a deduction which is fully met by the fact that the breath sounds are feeble and the expiration much prolonged; evidently the respiratory murmur cannot be distinct when a decreased amount of inspired air can enter the already filled vesicles, and when that which is expired has so little alveolar reactive tension left to create any prompt, brief, or sharply audible current in passing.

The *diagnosis* of *hypertrophic emphysema* of the lungs is established also by the characteristic border features, especially the extended and yet almost immobile lung boundaries, and the effects of their encroachment upon the areas of dulness of the heart, liver, and spleen.

DIFFERENTIAL DIAGNOSIS.—*Pneumothorax* is the only condition that may simulate emphysema to any appreciable extent, but, in the first place, the former is apt to be sudden and abrupt rather than gradual and long continued in its development, as in the case of emphysema. Then, again, pneumothorax is unilateral; the enlargement is one-sided, and the affected side hardly moves at all. The thorax is also generally small and emaciated. The unaffected side moves with exaggerated activity. The percussion note is more often tympanitic than simply hyperresonant, and is more apt to displace the heart (in a direction opposite to pressure) than is emphysema, which encroaches over the adjacent organs. Whereas in emphysema feeble breathing is heard, associated with whistling or fine bubbling râles, in pneumothorax the breath sounds are either absent over the affected side, or distant amphoric breathing, possibly with metallic tinkling, is detected. The coin-percussion test may reveal the metallic or bell sound peculiar to pneumothorax. And, finally, a zone of dulness at the base posterolaterally, the upper limit changing with the posture of the patient, may indicate the presence of liquid (hydropneumothorax).

It is highly important that other conditions than emphysema, which may be partially hidden by the enlarged and extended lungs, should be sought for in most cases. Thus aortic aneurisms, mediastinal tumors, cardiac, hepatic, and splenic enlargements, pulmonary tuberculosis, and bronchiectatic cavities, for example, may be overlooked in consequence.

SECTION III

THE HEART AND PERICARDIUM

CHAPTER IX

INSPECTION

Preliminary.—There is no organ of the chest or abdomen the anatomic and functional abnormalities of which lend themselves so readily and accurately to diagnosis by means of the physical methods of examination as the heart. The physical diagnosis of cardiac affections is also the most important, in an individual or exclusive sense, in that the physical signs of themselves are essential to a precise knowledge of the lesions. Furthermore, given the signs of a case of organic valvular disease, for example, and the location and character of the disorder, with its consequences upon the pulmonary and systemic circulations, especially in the way of engorgements, may be deduced by an Aristotelian logic with almost mathematical precision.

Before any satisfactory information concerning the diagnosis of cardiac diseases can be utilized, a review of the cardinal points of the clinical anatomy of the heart is of prime importance, since much depends upon this. Likewise the physiology of the heart has vital bearing upon the diagnosis of most of its affections, but this will be considered more appropriately under auscultation.

TOPOGRAPHIC AND RELATIONAL ANATOMY OF THE HEART AND ITS VALVES

Location of the Heart.—The heart being conoidal or irregularly pyramidal in shape, is so located between the anterior and posterior mediastini that its long axis is directed downward, forward, and to the left (at an angle of 60 degrees with that of the body), its apex behind the fifth interspace, ½ to 1 in. inside the left nipple line, the base directed upward, backward, and to the right side, its extreme border extending about 1 in. to the right of the right sternal line (fourth interspace). Its lower surface rests upon the central tendon of the diaphragm. Thus two-thirds of the organ, sometimes three-fourths, is on the left side. It is held from rolling about in the chest by its attachments to the great vessels at the base, and by the pericardial sac which envelops it firmly around the roots of the vessels, loosely below, leaving some space between the heart (right ventricle) and its diaphragmatic and sternal attachments analogous to the complementary pleural sinus.

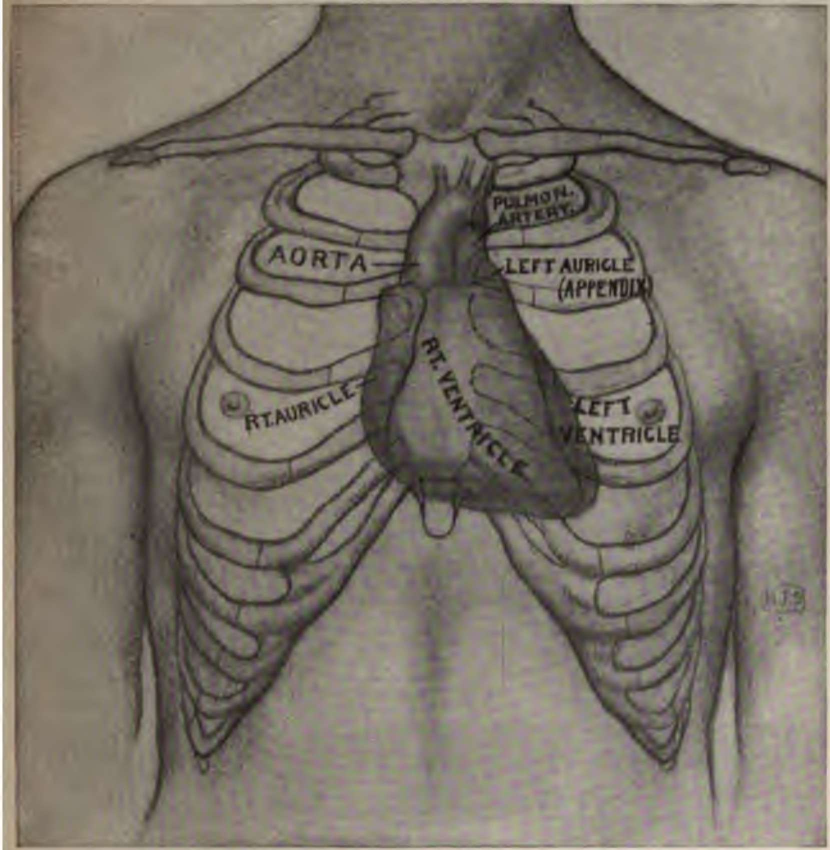


FIG. 42.—THE ANTERIOR ASPECT OF THE NORMAL HEART AND GREAT VESSELS, SHOWING THEIR RELATIONS TO THE ANATOMIC LANDMARKS (RIBS, STERNUM, CLAVICLES) OF THE FRONT OF THE THORAX. (Butler.)

The *relations of the anterior surface* of the heart to the chest wall in front are of most practical importance. This slightly curved surface is nearly parallel to, and in apposition with, the sternum and ribs, except where the lungs overlap by their anterior borders, and is triangular in shape. It includes the right appendix and most of the right auricle to the right of the midsternal line, the greater part of the right ventricle (a portion uncovered by lung), and small portions of the left appendix and left ventricle to the left of the same line.

Topographic Outline of the Heart on the Chest Wall.—Assuming the examiner's eyes to be on a level with the center of the heart, approximately—that is, opposite the left border of the sternum in the fourth interspace—the surface landmarks of the borders of the average normal heart may be projected as follows:

(1) *Upper Border.*—This may be defined by a horizontal line drawn through the upper edge of the third costal cartilages, extending from a point 1 in. to the left of the left sternal margin to a point ½ in. to the right of the right sternochondral articulation. This defines the highest point of the heart—the base—above which are the great vessels, and below which run the right and left boundaries, more or less outwardly.

(2) *Right Border.*—This is traced by a line curving outward slightly from the right upper point, just noted, down to the lower border of the fifth rib, nearly 1 in. to the right of the edge of the breast-bone. From thence, curving sharply inward, begins the

(3) *Lower Border.*—It slopes downward slightly toward the left, crossing the midsternal line at the junction of the upper and middle thirds of the xiphoid process, the sixth costal cartilage (left) near the sternal junction, and terminates in the fifth interspace near the apex, about 1 in. within the midclavicular or mammillary line.

(4) *Left, or Outer, Border.*—This corresponds with an oblique, slightly curved line, with its convexity outward, extending from the left end of the upper boundary (1 or 1½ in. to the left of the left sternal articulation of the third costal cartilage) to the apex in the fifth interspace, about 3½ in. from the midsternal line.

The *highest point* of the heart, the left auricle, is at the lower border of the sternal insertion of the second rib. The ***upper boundary*** or base line is the dividing line between the heart and the great vessels arising from it. The base line passes through the tops of the auricles.

The *right border* is formed by the right auricle (behind the sternum).

The *inferior border* is formed by the right ventricle and apical portion of the left ventricle, and is often spoken of as the *anatomic base* of the heart, in contradistinction to the commonly accepted *clinical base*, with its great vascular attachments.

The *left border* is formed exclusively by the deeper-seated left ventricle.

Posteriorly, the base of the heart corresponds with the level of the fifth or sixth dorsal vertebra, the apex with the eighth vertebra.

According to Broadbent, a diagonal line extending from the junction of the third costal cartilage with the left edge of the sternum downward to the seventh right chondrosternal articulation represents the usual position of the *auriculoventricular septum*.

Another line, passing downward from inside the third left costochondral articulation to a point about ½ in. within the apex limit, corresponds pretty closely with the course of the *interventricular septum*.

The *relations which the different parts of the heart bear to the chest wall* need to be considered also.

Beginning above, the roots of the great vessels lie directly behind the sternum, near the junction of the third left costal cartilage, the origin of the pulmonary artery being almost directly in front of that of the aorta.

Viewing the uncovered heart *in situ*, one discovers the lower two-thirds of the *right auricle* to the right of the sternum, the upper third behind that bone. The *left auricle* is hidden behind the right, except at its tip, which may be seen in the second left interspace close to the sternal edge. The *right ventricle* occupies all of the remaining triangular space anteriorly, partly behind the sternum and partly to the left of it, the narrow margin of the *left ventricle* projecting from behind the right at its left border, and terminating forward at the apex.

Positions of the Great Vessels.—The *pulmonary artery* arises at the level of the third left sternochondral junction, passes upward close to the left edge cf the sternum for nearly 2 in., to end behind the second left costal cartilage, where it bifurcates. The *aorta* (ascending) begins at the level of the third left chondrosternal articulation, and takes a course diagonally upward across the breast-bone to the second right chondrosternal articulation, projecting somewhat into the second interspace. The *arch of the aorta* lies back of the sternum, its transverse portion crossing at the level of the first intercostal space; posteriorly, its highest part reaches the level of the third dorsal vertebra. The aorta reaches the spine at the fourth dorsal vertebra, at which level the pulmonary artery and trachea both bifurcate.

The *innominate artery* arises opposite the first intercostal space behind the left half of the sternum. It ascends obliquely to the right sternoclavicular articulation, where it divides into the right common carotid and subclavian arteries.

The *superior vena cava* lies to the right of the aortic arch, about ½ in. to the right of the right sternal border, passing downward from the first costal cartilage to the second intercostal space.

Relations of the Heart to the Lungs.—The whole of the anterior surface of the heart is overlapped by lung, except that irregularly triangular, almost quadrilateral space corresponding to the lower portion of the right ventricle, due to the indentation of the anterior edge of the left lung. It may be well to recall the relations of the lung margins to the enveloped heart. The anterior borders of the lungs, descending from the apices, approach each other until they almost meet behind the middle of the sternum at the level of the second costal cartilage, whence they descend, closely apposed to each other, to the fourth chondral level. At this point they diverge, the right lung border passing gradually downward and outward, to cross the fifth cartilage and interspace, and meet the inferior border at the sixth rib in the midclavicular line; the left, turning abruptly outward at the fourth chondral level, passes along the lower margin of the same, obliquely crossing the fourth interspace near the parasternal line (almost corresponding to the costochondral junction), then turning slightly inward, like a reversed letter *s*, and again downward and outward across the fifth rib and interspace, unites with the inferior border at the sixth rib as on the right side.

Thus the boundaries of this superficial portion of the heart are as follows: the upper, at the lower border of the left fourth rib; the right, between the middle line and the left sternal line; the left, a little outside the left parasternal line, below the fifth interspace, where the heart overlaps the left lobe of the liver. It is this portion of the heart (right ventricle) in contact with the chest wall which, on percussion, gives rise to the so-called *area of absolute or superficial cardiac dulness.* Should the lung here completely fill the complementary pleural sinus, this relatively exposed region of the heart would be much smaller than the approximately 2½ × 2½ in. area.

Positions of the Valves.—These are pretty close together, topographically, bunched, as it were, near the junctions of the third and fourth ribs (left) and the third interspace with the sternum. The pulmonary valve point is the most superficial and superior, the tricuspid the most inferior, the aortic the most central, and the mitral the most outer and internal.

More precisely, the valves may be located according to the following topography. The *mitral* valve, the most deeply seated of all, is behind the left half of the sternum, on a level with the third interspace, fourth cartilage, and fourth interspace.

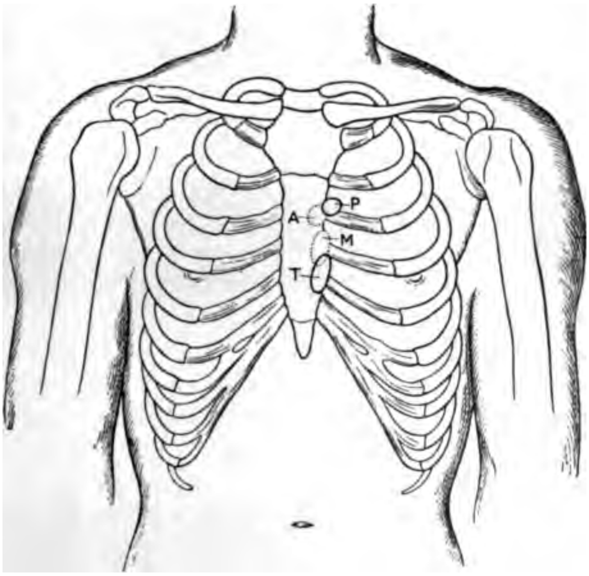


FIG. 43.—POSITION OF THE CARDIAC ORIFICES IN RELATION TO THE SURFACE OF THE CHEST. (Gibson and Russell.)

The *tricuspid*, the other *auriculoventricular valve*, lowest in situation, is behind the middle of the lower fourth of the sternum, corresponding to the fourth and fifth cartilages and the fourth interspace.

The *aortic valve*, most centrally located, lies behind the left half of the breast-bone, on a level with the third interspace and the lower portion of the third costal cartilage.

The *pulmonary valve* lies in front of part of the aortic, but a little higher and more to the left; its level is the third cartilage, and a needle pushed horizontally through the third left chondrosternal junction would about penetrate the middle of the pulmonary orifice.

A stethoscope, with a chest piece a little over 1 in. in diameter, placed with its outer edge coinciding with the left sternal margin and its upper and diametrically lower limits corresponding to the levels of the middle of the third and fourth costal cartilages respectively, will include a portion of all four valves. But, as will be pointed out later, under auscultation these anatomic surface landmarks do not correspond to the areas at which the sounds produced by valve closure are usually best heard.

Furthermore, the chest outlines of the heart vary somewhat in health owing to its normal *mobility*, swinging as it may from right to left, up and down, forward and backward, from its base with vascular attachments. This may result from *posture* and from *respiratory movement*, the changes affecting the outer border and apex principally. The left lateral and dorsal positions are most apt to alter the relation of the heart to the external landmarks. The movements, upward and downward, of the ribs and diaphragm rhythmically affect the upper and lower boundary relations only with forced respiration, for which due allowance may be made.

Age is a factor, also, in that with children the heart is about one rib higher, and has a trifle more surface in contact with the chest wall because of less volume of overlapping lung proportionately. On the other hand, in the aged the heart moves lower down, so that its inferior limit corresponds with the lower border of the sixth rib, or even the sixth interspace. On account of the increased volume of lung, however, a smaller portion of the heart is parietal, proportionately, than during the early and later middle years of life.

The Precordium.—From the preceding data of the clinical anatomy of the heart, it will appear that practically all of the physical signs referable to diseases of this organ occur within a limited, rather definite, area of the chest wall—the *precordium.* This precordial space, for ordinary purposes, constitutes a somewhat arbitrary rectangular area whose *upper boundary* is the second rib; its *inner boundary*, a vertical line 1 in. to the right of the right border of the sternum; its *outer boundary*, a vertical line coinciding with the left midclavicular or nipple line; its *lower boundary*, parallel with the horizontal upper, and passing through the sixth rib at the level of intersection with the preceding or mammillary line.

METHOD OF INSPECTION OF THE HEART

As in the examination of the chest, as a whole, with a view to ascertaining the physical signs of pulmonary diseases, so here it is essential for satisfactory results that the whole precordial and surrounding area be exposed to vision under both direct and oblique light—preferably daylight. For accuracy, the patient should be examined in both the sitting or standing and semirecumbent positions.

In the inspection of the precordium we observe this region as a whole; then any of its individual constituent features. The normal chest shows the slight flattening over the sternal area, and a symmetrical fulness of the two sides and of the rhythmical respiratory rise and fall; between the fifth and sixth ribs, about 2 in. from the left edge of the breast-bone, a slight thrusting movement, hardly more than ½ in. in diameter, is visible. These impulses average about four to one respiratory movement. In some healthy individuals the precordium is a trifle fuller than the corresponding region on the opposite side, occasionally due to greater muscular development in a left-handed person. Just as often, perhaps, if not more so, this area shows a little flatter aspect than on the right side. In many, also, a slight pulsation may be seen in the epigastrium, just below the tip of the ensiform cartilage, especially after exertion or during a certain amount of mental activity or emotional excitement.

We study: (1) *Abnormal prominence* or *depression* of the precordium; (2) *abnormal pulsations* within or near the borders of this space; (3) most important of all, the *apex-beat*.

(1) **The Precordium and its Adjacent Areas in General.**—(*a*) UNDUE PROMINENCE.—This is the most common change in the form. Allowance must be made for alterations not cardiac but costal or inflammatory in origin. Thus, such malformations as may be associated with spinal curvature, rickets, fractures, bony and cartilaginous hypertrophies, and tumors and the like must be differentiated. The same is true with regard to the unilateral or local bulgings due to pulmonary or pleural diseases.

Of strictly cardiac causation, the most common source of abnormal precordial fulness is enlargement of the organ—that condition of combined thickening of the walls and increase of chamber capacity known as ***hypertrophic dilation*** of the heart. The degree of bulging is usually quite moderate, and distinctly more noticeable in childhood with its thinner, yielding thoracic walls. In adults, especially those past middle life, only the greatest enlargements of the heart cause marked precordial bulging, provided, as a rule, that the condition is one of long standing. A moderate or slight bulging, therefore, limited to the lower half of the precordial space, but unaccompanied with a filling out of the contained interspaces, occurring especially in a person who has not reached middle life and who complains of breathlessness on a little exertion, is in itself significant of the probability of cardiac hypertrophy due to some valvular lesion.

When the alteration of the form of the precordium is more general, the prominence more marked, and unmistakable fulness of the intercostal spaces also evident, it points to the presence of a *pericardial effusion.* Of course, this precordial swelling is noticeable not so much as an independent as a comparative sign—the fact that it does not extend beyond the sternum, to the right; that it is usually seen farther to the left—beyond the nipple line; farther down—to the sixth interspace at times; and higher—to the third, even second interspace in severe cases, than where enlargement of the heart is the cause. Also, the younger the patient the more prominent the effect of a pericardial effusion upon the precordial shape.

Local bulgings above the fourth rib are almost invariably aneurismal. It is considered quite characteristic of aneurism of the innominate artery to find a rounded swelling in the suprasternal notch and behind the right sternoclavicular articulation, the sternal end of the collar-bone being even dislocated forward in some cases, and pulsating. Bulging in the first and second right interspaces near the sternal margin, or of the manubrium and second and third costal cartilages on both sides, indicates pressure and erosion from aneurism either of the ascending or transverse aortic arch.

(*b*) ABNORMAL DEPRESSION OF THE PRECORDIUM is sometimes seen. Aside from the collateral effect of scoliotic or rachitic deformities of the thorax, and of unilateral chronic pleural adhesions with or without an old, left-sided empyema, a portion of the precordium may be sunken because of *adherent pericardium* following chronic pericarditis. The superjacent pleura may also be attached to the region of adhesion of the visceral and parietal layers of the pericardium, thus aggravating the retracted area.

(2) **Abnormal Precordial Pulsations.**—Apart from the normal and abnormal apex-beat (to be considered under the next article), all other precordial pulsations visible (or palpable) are adventitious—abnormal—and more or less significant of cardiac derangement, aortic disease (aneurism), or adjacent pleuropulmonary disease.

(*a*) AT THE BASE AND BORDERS OF THE HEART.—Pulsations here may indicate aortic aneurism, enlargement of the heart (auricles), or exposure of the aorta and pulmonary artery from shrinking of the lungs due to fibroid phthisis or chronic pleurisy with adhesions.

Many of these pulsations are more evident when viewed at an angle, as from the side of the patient's bed. The natural visibility of new pulsations and their rhythmical relation to the apical impulse may be determined and confirmed with greater accuracy by such available artificial means as the following: Balfour attaches a short bristle carrying a tiny paper flag, by means of a pellet of beeswax, to the skin over the pulsation whose time in the cardiac cycle (systole or diastole) is to be determined, and a similar flag is placed over the apex-beat; the consequent exaggeration of these pulsations in the movements of the flag extremities enables the eye to detect more positively the slight or doubtful new pulsations, as well as to discriminate any difference in time in the movements of the flags. Another method, simpler and readier, is to pull bits of absorbent cotton into slender cones 2 or 3 in. long, then apply their bases to the skin over the pulsating spots, previously touched with mucilage or thick ointment (Butler). The expansile (aneurismal) character of a pulsation may also be noted by placing indicators on diametrically opposite sides of the pulsating tumor, and observing the divergence and convergence of their tips with expansion and contraction respectively.

As the base of the heart is completely covered by lung, as it moves backward with each systole, and as its impulse is transmitted with difficulty, also, because of the resistance and thickness of the ribs (including the pectoral muscles and adipose tissue) in that region, any *base beat* here—that is, practically above the level of the fourth rib—must mean either retraction of the lungs, enlargement of one or more heart chambers, especially in children with thin, elastic chest walls, or some dynamic or aneurismal affection of the great arteries.

The pathologic significance of pulsations occurring in parts other than the apical region of the heart may now be dealt with seriatim.

In the *suprasternal notch*, abnormal pulsations may be due to aneurism either of the innominate artery or of the transverse portion of the arch of the aorta. I have witnessed it also in chlorotic women with soft, flabby arterial walls and slow, sluggish heart action.

Pulsation in the *right first or second intercostal space*, near the sternal border, if expansile, systolic in time, and accompanied with some swelling, perhaps, is indicative of aneurism of the ascending portion of the aortic arch. If the expansile character of the pulsation is absent, the movement may be due either to transmitted violent cardiac action or to exposure of the aorta in cases where the heart is drawn to the right by right-sided pleuritic adhesions with retracted lung.

Pulsation in the *right second, third, and fourth interspaces* may be partly due to the last-named condition or to throbbing of the right auricle (presystolic in time), which is usually dilated because of secondary tricuspid regurgitation in such instances.

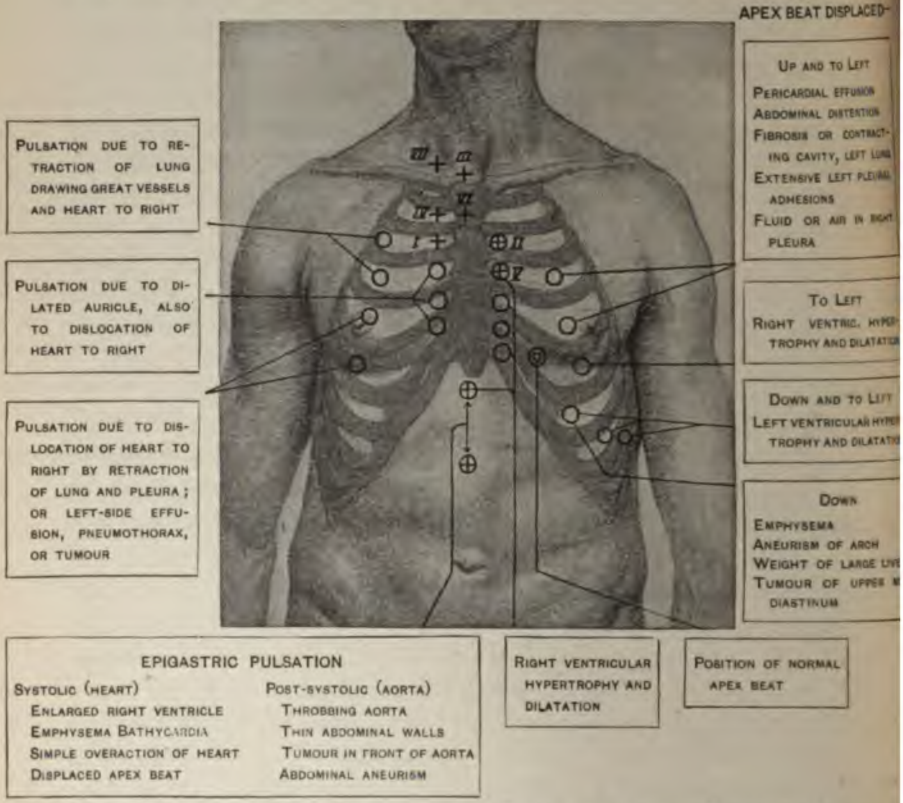


FIG. 44.—SHOWING THE INDICATIONS TO BE OBTAINED FROM THE POSITION OF THE APEX-BEAT AND OTHER THORACIC AND EPIGASTRIC PULSATIONS. *I, II, III, IV, V, VI* = pulsations of aortic aneurism (arch), numbered in order of frequency of occurrence. *VII* = innominate aneurism. *II* = also pulsation of pulmonary artery (and left auricular appendix?). *V* = also pulsation of right ventricle (and left auricular appendix?). ○ = cardiac impulses. + = aneurismal pulsations. ⊕ = both. (Butler.)

Pulsations in the *right third, fourth, and fifth interspaces,* near the right parasternal or midclavicular lines, are the result of displaced heart to the right, either from the pulling forces of right-sided pleural and pulmonary adhesion and retraction or the pushing effects of a large liquid pleural effusion, or of a pneumothorax on the left side.

Pulsation over the *manubrium* indicates eroding aneurism of the innominate artery, or of the transverse portion of the arch of the aorta.

Pulsations detected in the *left second or third interspace*, close to the margin of the breast-bone, may be arterial (pulmonary), aneurismal (aortic), or auricular (left). In the two first cases the pulsations are synchronous with or follow slightly the apex-beat; in the third they precede the apex-beat a trifle. If there are simultaneous physical signs of shrinking of the left lung, or of enlargement of the heart, the pulsation is probably due, in the first instance, to its uncovered contact with the walls of the interspaces, or to its ready transmission through condensed lung structure overlying, and, in the second instance, to a markedly dilated and engorged left auricle or conus arteriosus of the pulmonary artery, as is found in mitral stenosis. Aneurismal pulsation in this region would mean, obviously, involvement of the descending aorta.

Pulsations in the *third, fourth, and fifth interspaces*, between the left edge of the sternum and the left parasternal line, are due to hypertrophic dilation of the right ventricle, or to the very rare aneurism of the ventricular wall.

Pulsations in the *third and fourth interspaces*, in or slightly outside the *left midclavicular line*, usually mean a displaced apex-beat.

Occasionally a double impulse, accompanying with more or less regularity each systole, has been observed (Guttmann, Leyden, and others). It occurs in aggravated cases of mitral insufficiency, and arises from the non-coincidence of the contractions of the ventricles. The pulsation in the carotid artery corresponds with the first of these strokes only.

Extent.—The wider the area of impulse over the precordium—that is, one diffused broadly in two or three interspaces—the greater, as a rule, the hypertrophy of the heart. Unless the heart is displaced to the left, however, the pulsations do not extend beyond the left mammillary line. In cases where the chest wall is very thin and flexible, the pulsation may be quite diffused even with a normal size of the heart, particularly if its action is excited and the overlying lung thin or retracted.

(*b*) Systolic Retraction.—Before taking up the extracardiac border-line pulsations, this precordial, rhythmical *sinking in*, instead of thrusting out, may be considered. It affects usually the third, fourth, and fifth interspaces on the left side, over the body of the heart, around the apex-beat, and the left xiphochondral angular space. Its significance is *adhesive pericarditis.*

(*c*) Broadbent's Sign.—A systolic recession of the tenth and eleventh interspaces, below the inferior angle of the scapula, was described by Sir William Broadbent as a sign of adherent pericardium. It is seen in thin or emaciated individuals. It is explained as the result of a drawing upon the diaphragm by a hypertrophied and vigorously acting heart. It is conceivably an exaggeration of the normal but invisible slight pull which the heart makes during systole upon the central tendon of the diaphragm. Broadbent's sign is not limited to an indication of extensive pericardial adhesions, however, as it is sometimes present in cases of marked cardiac hypertrophy without the least suspicion or evidence of these.

Pulsation near the left border of the heart may be due to a "pulsating empyema" (*empyema pulsans*) lying close thereto, the probably tense collection of pus transmitting the heart's impulse to the adjacent interspaces.

Similarly, a perceptible pulsation may be seen in this region in certain cases of malignant vascular tumor of the lung.

Again, hepatized lung, as in lobar pneumonia, may convey to the surface of the chest, in the neighborhood of the heart, visible fine pulsatory movements or thrills wherever it may happen to be in intimate contact with the latter organ.

(*d*) Epigastric Pulsation.—Very important is the occurrence of abnormal pulsation in this extracardiac region, bounded by the costochondral sides of the epigastric angle forming at the xiphoid cartilage. A throbbing pulsation in the epigastrium may, however, be the simple, temporary, physiologic result of emotional excitement or sharp physical exercise, as a run up a flight of stairs just previous to examination. Pathologic epigastric pulsations have as more or less probable causes the appended conditions: cardiac, vascular, hepatic, neoplastic, neurotic.

In the first place, when epigastric pulsation is associated with absence of the apex-beat in its normal position, the possibility of displacement of the heart should be confirmed by the other methods of examination.

Hypertrophy and dilation of the right ventricle is the most common cardiac cause of epigastric pulsation. In such cases the impulse is systolic in time. The left ventricle being pushed back by the enlarged right ventricle, the latter communicates its pulsation to the lower part of the sternum, and to the left lobe of the liver where it lies against the tissues of the wall. This epigastric pulsation from dilated right ventricle may be due to emphysema, the heart being displaced laterally toward the breast-bone, and downward upon the diaphragm, both because of the crowding effect of the dilated lung and of the increased bulging and weight of its own enlargement, the strain upon the right ventricle in emphysema, on account of the diminished caliber of the pulmonary capillaries, being a well-known pathologic fact.

In hypertrophic dilation of the right ventricle, also, secondary to obstruction or incompetency of the mitral valve, particularly when the tricuspid orifice leaks because of the extreme dilation, epigastric pulsation is commonly noticed. This is augmented by a *pulsating liver* in the same region, the systolic impulse of the regurgitation through the vena cava inferior being transmitted also through the engorged, swollen liver of secondary venous congestion.

Epigastric pulsation may be caused by *aneurism of the abdominal aorta,* the expansile throb being transmitted through the left lobe of the liver; or, if the aneurism is a large, sacculated one, the pulsation may be conveyed direct to the surface from under the edge of this organ.

Frequently, in neurotic and neurasthenic individuals, especially in persons with comparatively empty abdomens, with thin, emaciated, or relaxed (as in women who have undergone several pregnancies) abdominal walls, an exaggerated aortic impulse in the epigastrium is plainly visible. A dynamic pulsation here may result, also, from hypertrophy of the left ventricle; from cardiac excitement, in anemia after hemorrhage, in young, hysteric females, and during the climacteric; in old persons of both sexes who may be arteriofibrotic or neurasthenic; and from the reflex effects of gastro-intestinal digestive disorders, movable kidney, etc.

The rhythm of the aneurismal and functional aortic epigastric pulsations is an instant later than the cardiac impulse (postsystolic).

Any *tumor* overlying the aorta in the epigastric region may communicate a pulsation. Thus, a pyloric cancer of the stomach, a pancreatic or hepatic growth, or enlarged lymph-glands (tubercular) may so act, except when the patient assumes the knee-chest position so as to allow the tumor to fall away from close contact with the aorta.

(*e*) A SYSTOLIC RECESSION of the epigastrium often accompanies chronic adhesive mediastino-pericarditis. Here the movement is the reverse of the ordinary epigastric pulsation—a systolic retraction instead of a systolic thrust outward. Sometimes the pericarditis has been so extensive that the combined epigastric and lower precordial pulsations produce an undulatory movement which may be confusing as to actual time of occurrence in the heart's cycle unless they are carefully and discriminatingly compared with the apex-beat, the epigastric and intercostal depressions and apical impulse, of course, being synchronous in systole.

(3) **The Apex-Beat.**—In studying the apex-beat we note the following points by inspection: (*a*) The *position* of the impulse; (*b*) its *extent*; (*c*) its *strength*; (*d*) its *rhythm*. The strength and rhythm of the apical impulse may be roughly estimated by the eye, but need to be verified, modified, and corrected by the employment of palpation.

These factors presuppose the visibility as well as tangibility of the apex-beat. But negative evidence as to the pulsation is not necessarily important from the clinical standpoint. For example, in very fat and muscular people the absence of the apex-beat may be quite consistent with the thick parietes. Again, the apex may happen to thrust behind a rib in a perfectly healthy individual with a variation of the anatomic relations.

(*a*) POSITION OF THE CARDIAC IMPULSE.—While the *normal position* of the cardiac impulse is commonly referred to as the apex-beat, it is actually the portion of the heart inside the true anatomic apex that strikes the chest wall. However, with each systole or contraction of the ventricles, a pulsation representing the outermost and lowermost point of discernible cardiac movement is seen normally in the fifth interspace, about ½ in. inside the left midclavicular or nipple line, or about 2½ to 3 in. from midsternum. This rhythmical impulse is confined to the same interspace, its lateral extent being usually less than 1 in. The character of the pulsation is simply a gentle outward movement and recession, having a regular rhythm corresponding in time with the carotid pulse.

There are several *normal variations* in the location of the apex-beat. Thus, in children under six years of age it is found behind the fifth rib, or in the fourth interspace; in the very old it pulsates in the sixth interspace, and often relatively nearer the median line than in the young. In persons with short, broad thoraces, also, the cardiac impulse may be seen in the fourth interspace; in the sixth in those with long, narrow chests.

Posture affects the position of the pulsation, in that lying on the left side causes it to be seen toward the axilla, from 1 to 2 in. to the left. The recumbent position renders the beat less visible than the upright. A right-sided shift of the impulse, although not so marked, can be observed when the individual lies upon the right side.

Respiratory movement modifies the position of the apex-beat. During a full inspiration one can notice the impulse lowered an interspace, even producing an epigastric pulsation; with the expiratory rise of the diaphragm it again appears above the sixth rib, and, if the effort is forced, slightly to the left as well.

Distention of the stomach after a full meal, or marked flatulence, pregnancy also, may cause an upward movement of the apex-beat.

The *pathologic causes* of displacement of the apex-beat may be summed up beforehand as *cardiac, pericardial,* or *external.* The *direct physical causes* are also three, in themselves the result of the operation of the preceding; these are *enlargement of the heart, dislocation of the heart,* and *deformity of the chest.* Apical displacements may be classified conveniently according to their direction, whether upward or downward, to the right or left.

(1) *Upward, or Upward and to the Left.*—The commonest causes of upward displacement of the apex-beat are intra-abdominal enlargements. These may include *ascites* (abdominal dropsy); *meteorism,* as from peritonitis; *large abdominal tumors* of various kinds. In these conditions the apex is pushed upward, and in extreme instances even slightly to the left at the same time. In the absence of abdominal distention, an apical impulse found in the fourth or third interspace, and more decidedly to the left, associated with general precordial fulness, is due to *pericardial effusion.* Although the dislocation is also higher than from abdominal causes, the impulse is, however, less distinctly visible because of the interposed liquid.

An upward pulling force upon the heart sometimes occurs from retraction of the upper lobe of the left lung affected with fibroid phthisis. In deformity of the thorax produced by spinal curvature, as from scoliosis or kyphosis, we may find the apex-beat in the fourth interspace.

(2) *Downward, or Downward and to the Left.*—Undoubtedly the variety of displacement most frequently encountered is that of *hypertrophy and dilation of the heart,* especially of the *left ventricle*—simultaneous projection down and to the left. The more the dis-

placement downward rather than to the left the greater the predominance of left than right ventricular enlargement, and of hypertrophy over dilation, particularly when the apical impulse is apparently forcible and circumscribed. The latter may be detected in the sixth interspace, ½ to 2 in. outside the midclavicular line, or in exceptionally extreme cases as far as the eighth interspace and in the midaxillary line.

Simple downward dislocation of the apex-beat may also be caused by *aneurism* of the aortic arch, pressure of *mediastinal solid growths, thoracic deformity, marked emphysema,* and downward traction upon the central tendon of the diaphragm by an *enlarged liver*; sometimes by moderate-sized pleural effusion on the left side, or a pyopneumothorax—to less degree.

(3) *To the Left.*—Displacements to the left which are simultaneously downward or upward have already been considered. There remain those which are practically horizontal in direction. Pleural conditions which, on the right side, push the heart to the left, or which, on the left side, pull it to the left, displace the apex-beat accordingly. Thus, a *pleuritic effusion* or *pneumothorax* on the *right* side, or the retraction of extensive *pleuritic adhesions,* with or without *chronic fibroid contraction* of the *left* lung, may so act respectively. Another, a cardiac cause, is often met with in displacement of the apex-beat merely to the left, namely, *hypertrophy* and *dilation* of the *right ventricle*; the apex-beat is found usually in, or very slightly outside, the mammillary line, in the fifth interspace.

(4) *To the Right.*—Displacement of the apex-beat may be caused by a reversal of the thoracic conditions mentioned in the preceding paragraph. Thus, *left-sided pleural effusion* or *pneumothorax,* or *right-sided adhesive pleurisy* and *fibroid contractions* of the *lung* produce such displacement. The apex-beat may be discovered in the epigastric region, in the angles formed by the ensiform cartilage and the right and left rib borders. In extreme cases it may be seen in the fourth and fifth interspaces, at the right edge of the sternum. At the same time, pulsation may be noticed as far to the right as the nipple line, but this is referable to the right auricle and ventricle.

Besides these affections, *deformity of the chest* and the rare and interesting congenital transposition of the viscera (*situs inversus*) may cause a *dextrocardia.* In the latter case the apex-beat may pulsate on the right side, in the region corresponding to its normal location upon the left.

To *summarize,* the principal causes of displaced apex-beat are:

Hypertrophy and dilation of the heart, down and to the left.

Pericardial effusion, up and to the left.

Chronic pleural and phthisical affections, right or left.

Emphysema, down and sometimes to the right.

Pressure of subdiaphragmatic conditions, up and sometimes to the left.

Pressure of aneurism or mediastinal growths, downward.

(*b*) ABNORMAL EXTENT OF THE APICAL IMPULSE.—Alteration in the extent of the apical impulse needs to be discriminated carefully from an impulse near by, due to the body of the heart, as a hypertrophied right ventricle. In instances of the former, the wavy, cyclic, intercostal recession is usually found extending to the left of the actual apical thrust (as determined better by palpation), whereas, in the latter, the movement is confined within the anatomical limits of the heart; indeed, often to that area within the left parasternal line which is uncovered by lung. In marked downward and outward displacement of the apex, not infrequently a conjoined precordial pulsation, due to mass and apical movement of the heart, may be witnessed, as in cases of greatly enlarged heart (bovine heart, *cor bovis*), the result of prolonged regurgitation from insufficiency or leakage of the aortic valve.

A systolic retraction of the interspaces around the apex-beat should also not be confounded with mere increased area of apical pulsation. This does not mean that the apex retracts instead of thrusts forward with ventricular contraction, but that pericardial adhesions exist in such relation to the apex that they are drawn upon during systole, or that the sinking in of the interspaces is due to atmospheric pressure from without, induced by negative pressure within the chest because of the vigorous action of a heart in hypertrophic dilation.

The extent of apical impulse may be abnormal in three ways: it may be *increased*, *diminished*, or it may be *absent* totally.

(1) *Increased extent* of apex-beat is almost always associated with increased force, either actual or relative, and with displacement. As increased strength of beat can be estimated best by touch, and only inferred by sight, its relation to extent will necessarily include so much as can be gained by palpation as well as by inspection, if partial statements are to be avoided.

At the outset it may be set down that an impulse in the fifth interspace more than 1 in. (2½ cm.) in breadth is abnormal. A slight or moderate increase in the area of apical pulsation, in the fifth or sixth

or outside the left midclavicular line, indicates hyper-
ement of the heart, especially of the left ventricle. In
cases, where the impulse involves two or three inter-
the mammillary line—the fifth, sixth, and seventh,
a heaving character in the center, shading off to a fine,
ear the periphery of the movement, points to the massive
of hypertrophy over dilation, although both are in com-
he absence of a central thrust, however, and a still more
but evidently weaker, impulse, spreading outward rather than
ward, with slight in-and-out movement, indicates weakening of
eart muscle and dilation overcoming hypertrophy, at least temporarily. In other word [illegible] ndition exemplifies the fact
that the extent of the ap [illegible] ncreased without the strength
eing increased, and that t [illegible] r the extent the weaker is the
entricular muscle likely to be, since it rolls, glides, slaps, or flops against the chest wall in flabby fashion in dilation. The more circumscribed, although yet increased, extent of apical pulsation, due to predominant hypertrophy, on the contrary, manifests a more direct, concentrated, plunging thrust, as of a knuckle of a hand within the chest.

An apex-beat of moderate increase of extent and strength, without dislocation, is apparent in a group of disorders more or less functional in nature. Here may be mentioned *nervous palpitation,* as from emotional excitement; *exophthalmic goiter* (Basedow's or Graves's disease), and poisoning by *alcohol, tobacco, tea, coffee, strychnin,* etc.

A *relative increase* in extent of the apex-beat is found in cases of marked retraction of the lungs—anterior border. Here, also, there is no displacement of the position of the apical impulse unless there are at the same time extensive and firm pleuritic adhesions.

A wider area of apex pulsation may be seen as the result of simple dilation of the heart from the weakening of the ventricular walls affected with fatty degeneration, consequent upon severe acute febrile disease, chronic alcoholism, and the like.

It should be borne in mind, finally, that certain normal conditions may give rise to a comparatively increased extent of apical pulsation. Such may be—temporarily, of course—the posture, as when the individual leans forward; thin chest walls, as in children; physical exertion or mental excitement, and at the end of deep expiratory efforts. As, after violent physical exercise—athletic, occupational, or incidental to the modern rush for scheduled trains, and so on—a transient acute dilation of the heart may ensue, with consequent increased area of cardiac and apical impulse, with slight displacement of the latter to the left, a proper estimate as to the actual condition of the heart can be made only when it is examined at such a length of time thereafter as to exclude their possible modifying effects.

(2) *Diminished extent* or *absence* of apex-beat may be considered together. They may or may not be accompanied with weakened impulse. Provided that external conditions (to be mentioned below) are absent as factors, a diminished or just apparent apex pulsation usually indicates weak heart; in fact, it is palpably weakened.

In certain normal persons, it should be remembered, nevertheless, the apex-beat may be barely perceptible, or not at all so. This may occur especially in thick-chested or large-lunged or phlegmatic people, in the dorsal position, and sometimes at the height of a full inspiration.

Pathologically it is diminished or lost in the following:

Overlapping emphysematous lung.

Encroaching pericardial and pleuritic effusions.

Myocardial degeneration and dilation.

Shock, collapse, hemorrhage, acute febrile and chronic wasting disease.

Pericardial adhesions (replaced by systolic retraction).

Sometimes in stenosis of the aortic valve orifice.

Inflammatory or edematous tumefactions of the skin and mural tissues.

A moderate degree of emphysema may partially conceal the apical activity of a normal heart; a marked degree may, even with hypertrophic dilation of the right ventricle, entirely obliterate any impulse except in the upper part of the epigastrium. Absence of pulsation may be due, also, to sufficient downward dislocation of the apex to cause it to beat just behind the sixth rib.

Diminished area of impulse from pleural and pericardial effusions may be the result of the displacement as much as of the interposed liquid, and may be restored for the time by having the patient stoop forward.

Weak heart may be diagnosticated without difficulty whenever a previously increased extent of apex-beat in the fifth and sixth interspaces, outside the nipple line, gives place to diminished extent, and only in the fifth interspace.

A peculiar, almost paradoxical, physical sign is the diminished apical pulsation of certain cases of *aortic stenosis*, since the strain of this lesion produces decided hypertrophy of the left ventricle. It is explained by the obstructive delay and longer duration required by the ventricle to propel the blood through the orifice, so that the prompt apical thrust which would ordinarily occur is converted into a gradual, labored, prolonged twist of light contact with the chest wall comparatively.

(*c*) ABNORMAL STRENGTH OF THE CARDIAC IMPULSE.—Increase, diminution, or absence of apical pulsation usually corresponds directly with similar qualities of extent, although not invariably. The strength may be increased without an increase in the extent, and, *vice versa*, the extent may be increased and the strength actually diminished. As intimated before, the characteristics of apical force are most precisely determined by palpation, but to the trained observer the intercostal movements as indices of myocardial power are quite significant. Obviously, it is the amplitude or excursion of movement which is suggestive: the more circumscribed and bellied the rhythmical thrust the greater the firmness of cardiac muscle; the more flatly wavy and broadly fibrillary the action the weaker it is. Thus, also, a firm, moderate-sized hypertrophy of the left ventricle exemplifies the statement of increased force without increased extent of impulse, while the globularly enlarged heart of double-sided dilation, with more or less fatty degeneration, exemplifies the converse.

It should not be forgotten that many individuals, in good health and with sound hearts, manifest no apical impulse whatever. Also, that as in cases of increased extent of pulsation, so increased force may be due to purely psychic or neurotic causes, as in hysteria, Basedow's disease, insidious toxic and temperamental conditions, and the like.

(*d*) ABNORMALITIES OF RHYTHM.—Apart from mere increase or decrease in the frequency (denominated, respectively, *tachycardia* and *bradycardia*) of the apex-beats, the normal rhythm or regularity may be visibly disturbed as regards the time of their succession and the pauses between them, their fulness and force, both as single and variously grouped impulses.

Cardiac arhythmia may thus be noticed in connection with observation of the carotid pulse. The heart-beats may visibly alternate regularly—that is, every other beat fails to appear, or, at regular intervals, say every third or seventh or eleventh beat, drops; this constitutes the true *intermittent* heart, and may be normal and peculiar to the individual, or signify a transient functional disorder or permanent organic valvular or myocardial disease.

Where the intervals between the dropped beats do not occur at regular lengths, and where the beats that do appear succeed each other without regularity of force, frequency, extent, and interval, so that the cardiac impulse seems to be tumultuous and incoordinate, reeling and floundering like a drunken man, the arhythmia is denominated simply as *cardiac irregularity*. This phenomenon is always indicative of grave organic heart disease, and often of imminent and speedy dissolution.

As these and other special forms of arhythmia may be detected and estimated with greater accuracy by palpation, they will be discussed further under that head.

Rhythmic lateral displacement of the heart is not infrequently a valuable sign of *unilateral pleuritic exudate*, as first pointed out by C. L. Greene. It is most marked in moderate effusions; the heart approaches the affected side in inspiration, and moves outward during expiration; the extent of the movement is about 2 in., and it may be detected by fluorscopic examination, auscultatory percussion, and ordinary deep percussion, as well as by inspection of the apex-beat.

CHAPTER X

PALPATION

Palpation determines the physical signs discoverable by inspection, and so confirms its results; checks or modifies its results as regards certain dubious signs that may seem evident, but are really deceiving to the eye, and contributes certain other signs which cannot be considered visible. While supplementary to inspection, therefore, in the order of investigation of the cardiac phenomena in the precordium and its vicinity, palpation is so necessary to render complete and intelligible these phenomena that practically these two methods are most often employed simultaneously, the hand closely following the eye in nearly every detail before passing to the next physical sign, which receives the same conjoint study, and so on.

Thus palpation enlarges our knowledge as to the form of the precordial space; as to abnormal pulsations, even detecting those which are too feeble to be noticed by the eye; and determines the situation of the (anatomic) apical impulse, its force, extent, and character and rhythm, as well as the condition of the superficial tissues and intercostal spaces, especially in relation to the signs of edema and pus accumulations. In addition to these, palpation discovers the peculiar vibrations known as *thrills*, connected with organic valvular defects, and the presence of *pericardial friction fremitus.*

The visible and palpable phenomena of the arteries and veins are intimately connected with those of the heart, physiologically, pathologically, and clinically, and all must be considered together in physical diagnosis. Nevertheless, the arterial and venous signs will be dealt with in a subsequent chapter, so as to avoid confusion by reason of the extra facts superadded to those of strictly cardiac origin.

SHAPE OF THE PRECORDIUM

Although primarily and satisfactorily perceived by inspection, any undue general or local precordial prominence or depression may be more fully investigated by palpation. At the same time, some knowledge of the character and cause of the altered form may be derived by touch; as to whether the increased size, for example, is due to an exostosis or bony costal growth, an old fracture callus, a localized inflammatory edema, excessive muscular development, or to hypertrophy of the heart or pericardial effusion on account of increased sensation of resistance in the intercostal spaces in or beyond the lung-exposed area. Intercostal depressions are likewise recognizable as compared to the normal fulness and firmness of the corresponding spaces on the opposite side.

ABNORMAL PULSATIONS

Some that are not visible are palpable, and may seem to be extra apex-beats of feeble force, when not above the fourth rib. At the base of the heart, the pathologic pulsations noted under inspection are particularly amenable to palpation. Thus the aneurismal expansile pulsations, systolic in time, over the aortic arch may be felt; also the dynamic pulsations of the pulmonary artery and the auricles, either from exposure by retracted lung or because of hypertrophied and dilated right ventricle, and left auricle especially. The *diastolic shock* accompanying closure of the aortic valve in aortic aneurism may also be felt in this region.

Below the fourth rib, the epigastric pulsation due to hypertrophied right ventricle, or to pulsating liver (venous), or the heave over the body of this portion of the heart, between the sternal margin and the left parasternal line, are quite perceptible to touch.

It should be borne in mind just here that the hand of the examiner not infrequently detects a slight, diffuse shock over the middle of the precordium, even in health, and terminates in the fifth interspace in the more circumscribed, plunging throb of the apex-beat. This is more distinctly felt, of course, in those with thin chest walls, and especially when leaning slightly forward. In such persons, in such a posture, the hand placed in the third and fourth interspaces may also perceive a normal sharp jerk or shock during diastole, due to the closure of the two semilunar valves—aortic and pulmonary.

"When the whole heart is dilated and hypertrophied, a more or less violent shock may be felt over the entire cardiac area, and when the size of the heart is considerable, this shock may be double—the first forcible and systolic, the second less forcible and diastolic, the result of the rebound of the enlarged heart from the posterior thoracic walls" (Balfour).

THE APEX-BEAT

Position of the Impulse.—The *method* is important in ascertaining this point. A general or approximate knowledge as to the position of the apex-beat should be obtained first by applying the right hand lightly and evenly just below the left nipple, the examiner standing to the patient's right. In this way one gets a palpable survey of the region in which a displaced apex may possibly be found. At that portion of the hand under which the most distinct impulse is felt—as under the second knuckle, for example—the apex may roughly be estimated to be. Marking the spot by the eye, on removal of the hand the precise apical pulsation may then be localized by noting its point of maximum throb with the tips of one or two fingers. It is assumed that the patient is in the standing, sitting, or semi-reclining posture.

In the normal chest, the apical as well as the general cardiac impulse is better appreciated in those with thin walls, at the end of expiration, while the lungs are retracted as much as possible, and while the body leans forward. In cases of diffuse apex-beat, where, indeed, fine surface vibrations may be seen extending over an area wider than they are perceptible to the touch, accurate determination of the apical position may be baffled. But wherever within such a region the finger of the diagnostician is met apparently by another inside the chest rhythmically poking outward against it—in other words, at the most circumscribed point of most marked impulse—there the apex may be considered to functionate.

Palpation corroborates with exactness the displacements of the apex-beat due to the respective causes given under inspection. Since the apical impulse corresponds with the cardiac systole, and occurs a fraction of a second before the radial pulse, its importance as a standard for the timing of abnormal pulsations is obvious. It should be recollected, also, that the extreme left border of the apical impulse in the fifth interspace extends nearly an inch beyond the actual maximum throb of the apex.

Extent and Strength of Impulse.—The relative value in accuracy of palpation and inspection varies in respect to the width or extent of the apical pulsation; but, as intimated before, while the force of the apex-beat may only be inferred by inspection, its more precise estimation may be determined directly by palpation. As to the first point, then, in some cases the extent may be seen more widely than felt, while in others the area of impulse is tangible a little beyond any visible vibrations.

Before considering the principal pathologic indications of alterations of the strength and extent of the apical cardiac impulse, two things preliminary should be borne in mind, namely, the relation of these combined factors and their normal variations. In regard to the former, although to a certain degree increased force of apex-beat is accompanied with an increase of extent, this is not necessarily inversely so. Thus, most often a moderate increase of the extent usually means a predominantly hypertrophied heart; on the other hand, a marked increase in the area of pulsation is commonly palpable with marked weakness of the heart muscle—a predominance of dilation and flabbiness, with a rounded and enlarged apex.

In regard to the other point, it is important to remember that *age*, *posture*, *respiration*, *diet*, *physical exercise*, and *emotional excitement* may influence the force as well as partly the position of the apex-beat. Thus, in *childhood* the force of the beat is relatively greater, on account of the thin, flexible chest wall and the condition of the lungs. In *old age* it may be relatively weak because of thick chest walls and senile hypertrophy of the lungs, or stronger because of senile hypertrophy of the heart. Again, in the normal state, if the individual inclines his body slightly forward and to the *left*, the apex-beat becomes temporarily stronger, while if to the *right*, it grows faint or disappears, only the right ventricular impulse being felt near the sternum in the fourth and fifth interspaces, or in the upper epigastric region. At the *end of expiration*, particularly when the breath is held for a moment, the apical force is increased; also after a *full, stimulating meal*. Finally, the force and extent are increased during physiologically increased cardiac activity from more or less bodily exertion and psychic stress.

Palpable *exaggeration* of apical force is noted in connection with the abuse of tea, coffee, tobacco, alcohol, alcoholic and narcotic proprietary and "patent" medicines and other drugs used by *habitués*. In the early stages of acute fevers, in nervous palpitation, so-called, hysteria and other neuroses, and associated at times with the tachycardia of exophthalmic goiter, the same physical sign may be perceived.

The commonest abnormality is a forcible apex-beat of moderate increase in area felt over a manifestly displaced apex in the fifth interspace, a trifle outside the nipple line, or in the sixth and fifth interspaces, perhaps, as much as 1 or 1½ in. outside. This is always a clear indication of enlarged heart with hypertrophied walls. The greater the amount of associated impulse over the body of the heart, and of epigastric pulsation, the greater the size and massiveness of the ventricles.

The force and extent of the apex-beat are increased also in the earlier stages of *acute endocarditis* and *pericarditis*, by *pericardial adhesions*, and by such extrinsic causes as *chronic nephritis* (hypertrophy of left ventricle), *retraction of lung*, or *consolidation of lung* overlapping the apex.

Weakening or lessening of the apex-beat is, as pointed out under inspection, found in persons in health, although not infrequently an apex-beat that is invisible may be at least faintly palpable. In twofold generality, the causes of diminished strength of apical impulse are either those which weaken the myocardium or those which interpose some adventitious substance between the heart and the chest wall. These causes were detailed in treating of inspection. Suffice it here simply to mention, under the first of the preceding conditions, *myocarditis* and *fatty degeneration* due to endocarditis and pericarditis, or to prolonged and exhausting infectious fevers, anemias, cachexias, obesity, inanition, etc. Under the second category are to be placed *emphysema* and *pleural* and *pericardial effusions*.

Whenever fatness or undue thickness of the chest wall or emphysema of the lung can be excluded, and the signs of pericardial or pleural effusion are absent, diminished force of the apex-beat generally signifies cardiac weakness.

Total imperceptibility to touch, of the apex-beat, may, in some cases of cardiac or adjacent disease, be noted as having the usual problematic value of negative evidence. That is, in a stout person, for example, it may have been a characteristic before the onset of the malady; it may be simply a manifestation of marked general weakness from a protracted, prostrating illness; it may have the relative value indicative of emphysematous or pleuritic interference rather than any direct cardiac or pericarditic affection. Absent apical impulse due to myocardial troubles may result from an exaggeration of those conditions, causing lessened or weakened impulse. Not infrequently a previously imperceptible pulsation may become palpable after a little exertion on the part of the patient, such as sitting up in bed or, if his condition permits, walking to and fro rapidly for a dozen or twenty paces, or taking several deep, forcible breaths. The administration for a few days of cardiac stimulants may also elicit a palpable apex-beat.

It is well to repeat what was said under inspection concerning absence of the apex-beat in *stenosis of the commencement of the aorta,* even where there are percutory and auscultatory signs of hypertrophy of the left ventricle, the slow ventricular contraction from difficulty in emptying itself aborting the real apical thrust.

Systolic Recession.—The apex-beat is often wanting because replaced by this condition, which may be even more perceptible to the hand than to vision. Systolic retraction is seldom limited to the apical region, however, but is usually but a part of the rhythmical drawing in of the whole, lower portion of the heart area found in cases of pericardial or mediastinal adhesions. In a fair percentage of instances this sign is associated with that of Broadbent, namely, a similar rhythmical recession in the eleventh or twelfth left interspace posteriorly. According to Gibson, an important accompanying sign of adherent pericardium is a distinct impulse of "rebound or recoil" felt along with the diastole.

Rhythm of the Apical Impulse.—By palpation this may be determined with much greater accuracy than by inspection. This pertains to all forms of intermittency and irregularity, as pointed out under the latter heading. It should be remembered that inequalities of frequency, interval, size, and strength of the apex-beats are significant phenomena of failure of the cardiac muscle and function.

Groupings of the heart-beats in twos and threes may occur, corresponding to the analogous *bigeminal* and *trigeminal pulse.*

Doubling of the Apex-Beat.—By this is understood two pulsations of the apex to a single carotid pulse-beat, or radial. The phenomenon was attributed by Skoda, Leyden, and others to a *hemisystole*; that is, to an action of the heart in which the ventricles do not contract simultaneously. A more rational explanation of doubled apex-beat is the occurrence of alternating weak contractions of the heart, corresponding to alternating pulse (*pulsus alternans*), every other ventricular systole being too feeble to transmit a pulse-wave as far as the radial artery, but not so feeble as not to be palpable itself. Indeed, however, the alternating weak apical impulse is seldom so feeble that at least a weak pulsation over the carotid artery may not be felt.

ADVENTITIOUS PALPABLE SIGNS

Cardiac Thrills.—A *thrill* is a short, trembling sensation, felt usually in the region of the apex. It is of endocardial origin, the result of obstructive or incompetent orificial or valvular lesions, or, when felt at the base of the heart on the left side, may be due to the diminished plasticity of anemic blood. In any case, thrill is produced only at the time the blood is passing through the orifice affected; during systole, through the arterial—aortic and pulmonary—openings, and through the auraculoventricular—mitral and tricuspid—openings during diastole. The feel of a thrill simulating that of the purring of a cat led Laennec to call it *fremissement cataire*, and the Germans, *katzenschnürren*. The sensation communicated to the fingers may also be considered analogous to the tremor of a roughly working steam feed-pump, or marine engine.

As thrills usually coincide with the valvular *murmurs* heard on auscultation, a careful recognition of them may serve as preliminary or presumptive clues to the nature of the pathologic difficulty, whether an insufficiency due to degenerated valves, or a stenosis of the orifice, which causes the whirling eddies of the blood stream, whose vibrations become thus palpable and audible on the chest surface. As will be shown later, also, in treating of auscultation, where the murmurs have diagnostic significance according to certain points, so, in regard to thrills, it is necessary, *first*, to *locate* them; *secondly*, to *time* them—that is, to determine their rhythm; *thirdly*, to *circumscribe* them—that is, note their extent or area of diffusion. Most thrills occur at the selective mitral region, near the apex. Those most distinct are presystolic in rhythm, and the same involve a larger area of vibration than the systolic or diastolic. The principal thrills are as follows:

MITRAL PRESYSTOLIC THRILL.—At the *apex*, the commonest distinctive thrill is that which occurs just before the systolic impulse, and terminates abruptly at the height of impact of the latter. It has a rough, hesitating, purring, or jogging character. It is usually limited to a moderately diffuse area, rather more inside than outside the apex-beat, which is seldom displaced beyond the nipple line. In some exceptional cases the thrill occupies the greater part of diastole, commencing feebly, and reaching its maximum intensity immediately preceding the apical thrust—*diastolic thrill of mitral stenosis.* As indicated by the last word, this apical thrill signifies a narrowing at the mitral orifice, since the blood passes through that orifice during diastole, augmented at the end by the contraction of the left auricle, which thus develops the difficulty at that time (presystole) in causing the thrill.

MITRAL SYSTOLIC THRILL.—This is generally a soft, short, indistinct tremor, felt just over the apex, which may be outside the nipple line in the fifth or sixth interspace. It coincides with the lifting and receding of the apical impulse. It is due to leakage or insufficiency at the mitral orifice. This valve should be closed during systole; consequently the systolic thrill means a regurgitating whirling of opposing currents of blood permitted by the incompetency at that time.

Very rarely, delicate thrills may be palpable over the apical region which are transmitted downward from the aortic area, especially a systolic one due to aortic obstruction, and sometimes a diastolic one due to aortic regurgitation.

AORTIC SYSTOLIC THRILL.—This is most distinctly felt in the second right intercostal space, and occurs next in frequency to the mitral presystolic. It is often perceptible over the carotid, and along the right and left margins of the sternum, as far down as the ensiform, perhaps. As the aortic valve should be open during systole, a disturbance in the onflow of blood must be due to obstructive thickening or atheromatous stenosis of the orifice to produce a systolic thrill at its maximum in that location.

AORTIC DIASTOLIC THRILL.—This has its point of greatest intensity, which is rarely marked, however, also in the second right interspace. It is a soft, continuous thrill, felt along the sternum, toward the apex, during most of the diastolic time; even over most of the precordium in aggravated cases.

The thrill of aortic stenosis must not be confounded with that due to aneurism of the aortic arch. Here the peculiar whizzing sensation may be discovered between the suprasternal notch to the level of the fourth rib on either side of the sternum, and is usually accompanied with a pulsating swelling.

THRILLS DUE TO TRICUSPID AND PULMONARY VALVE LESIONS.—A *systolic* thrill felt over the lower part of the sternum is much more likely to be transmitted from the aortic orifice, and therefore, as the rare result of *tricuspid regurgitation*, can be assuredly diagnosed only when associated with synchronous *pulsation of the jugular veins*.

The other three forms of right-sided valvular disease analogous to the left-sided lesions referred to, although possibly attended with thrills, occur so rarely that they may practically be unheeded here. This is especially true of tricuspid stenosis (diastolic, presystolic) and pulmonary regurgitation (diastolic). In the second or third left interspace, a *systolic* thrill may be due to the congenital pulmonary stenosis, or to fibroid phthisis with retraction of the left lung at its anterior border, accompanied with exaggerated activity of the right ventricle. A little higher, in the first interspace, slight thrill may be felt in exophthalmic goiter.

Thrill in the second left intercostal space may be quite pronounced in another form of rare congenital heart disease, namely, permanent patency of the *ductus arteriosus*. This thrill lasts throughout systole and a part of diastole, owing to the fact that "the blood stream flows from the higher pressure of the aorta to the lower pressure of the pulmonary artery. It must therefore generally occur after the aortic pressure has reached a certain level, and will persist until it has fallen, at least to some extent" (Gibson).

Attempts have been made to obtain *cardiographic tracings* of thrills, but unsuccessfully. In cases where the cardiogram recorded any variation at all, it was due to interrupted or irregular heart action, and not to thrill, although that was easily palpable to light pressure of the fingers.

Pericardial Friction Fremitus.—This is a superficial rubbing sensation similar to that felt over a patch of dry pleuritis, and usually decidedly rougher than an endocardial thrill. I have described it to students as simulated by lightly passing a piece of emery paper across the hand. It is a rhythmic to-and-fro sensation—systolic and diastolic—felt over the body of the heart, and obviously signifies a dry, plastic, fibrinous pericarditis.

Fluctuation.—In instances of large, copious liquid effusions into the pericardial sac, waves of fluctuation may sometimes be felt in the lower precordial interspaces.

Valve Shocks.—These are palpable in certain cases because of the exaggerated intensity of closure of the semilunar and auriculoventricular sets of valves. Conditions affecting the former are distinctly felt in the upper precordium as diastolic shocks, and those of the latter producing systolic shocks, usually in the fourth intercostal space near the sternum—left edge—but only in persons with thin chest walls in both cases. Increased tension in the aorta or pulmonary artery, from left ventricular hypertrophy or increased peripheral resistance in the systemic arteries, or right ventricular hypertrophy and increased resistance in the pulmonary circulation, are indicated, respectively. Over the lower half of the precordium the systolic shock points to hypertrophic dilation of the ventricles.

CHAPTER XI

PERCUSSION

By this method of examination we may determine the *size, shape,* and *location* of the heart. These objects are attained essentially by delimiting the heart from the other thoracic, and from the abdominal, organs which border upon it. The fact that the heart is an airless organ makes it possible to outline its relations only with those adjacent structures which are more or less air-containing, and thus, on percussion, give rise to different qualities, intensities, etc., of sound.

As the heart is surrounded and partially overlapped on three sides by the lungs, the determination of the heart-lung boundary is by all odds practically the most important. The percutory relations of heart to stomach and to liver are seldom available for diagnostic purposes. Obviously, in outlining that portion of the heart uncovered by lung, one at the same time determines any extension or retraction of the anterior border of the left lung.

Percussion of the precordium develops the presence of two areas or varieties of dulness, according as we percuss directly over that small portion of the heart uncovered by lung and in close contact with the chest wall, or over a surrounding zone of the heart overlapped by a portion of the anterior lung margins. The first is known as the *area of absolute or superficial cardiac dulness*; the second, as the *area of relative or deep cardiac dulness.* With good reason, so as to avoid confusion and reversal of terms—a common error and difficulty among medical students—Butler prefers the following designations: the *exposed dulness,* the *covered dulness,* and, both together, the *entire dulness* of the heart.

PERCUSSION OF THE NORMAL HEART

The aim being principally, in clinical work, to ascertain the size of the heart in two dimensions, as projected in an approximate plane upon the chest surface, and to determine the cardiac relations to the overlying lung especially, as well as the presence or absence of any abnormal structure in contact with the heart and great vessels, the percussion of the normal heart areas of absolute and relative dulness needs first to be studied before the pathologic deviations can be estimated fairly.

As in the percussion of pulmonary conditions, so here much depends upon the uniformity and dexterity with which the particular method is applied, the accuracy and reliability of the technic, and the acuteness of perception of the physician.

Methods of Percussion.—There are three methods applicable to the determination of the areas of cardiac dulness: (1) Ordinary *pleximetric* percussion, with the fingers alone—at once the most serviceable and satisfactory—or with an artificial pleximeter; (2) *palpatory* percussion, and (3) *auscultatory* percussion. All three methods have distinctive value; the first has the advantage of directness, ordinarily sufficient accuracy, and practical convenience; the finger method enables the examiner also to note the sense of resistance. This latter is, on the other hand, the sole basis of the palpatory method, without the sound effects and differences, and, with the auscultatory method, constitutes an important addition to the more precise outlining of the heart requisite in cases needing special study because of complicating or obscuring physical conditions.

In the practise of ordinary finger percussion two facts should be borne in mind, namely, that only light strokes must be employed to detect the boundaries of the area of superficial or absolute dulness, strong, heavy strokes being necessary to elicit the deep or relative dulness. Before describing the technical details of the various methods of percussion, the student should be acquainted with the normal extent, shape, and limits of these two cardiac areas.

The *superficial or exposed cardiac dulness* is roughly quadrilateral in shape, or sometimes triangular. Its upper and outer boundaries correspond to the anterior border of the left lung. Superiorly, therefore, the absolutely dull or flat sound begins at the upper edge of the fourth left costal cartilage in the parasternal line. Thence the dulness continues vertically downward to the upper border of the sixth rib. The determination of this inferior boundary of the superficial dulness is a matter of *anatomic inference*, however, as it cannot possibly be separated from the liver dulness which meets it. This anatomic relationship may be facilitated in the delimitation by ascertaining the position of the apex-beat, a straight line from which, traced inward to the upper border of the liver dulness, may be considered a dividing line, all dulness above which, on the left side, is due to the heart. The outer boundary of absolute cardiac dulness passes obliquely downward and outward in an irregularly, slightly curved line, from the fourth rib to the sixth, between the parasternal and mammillary lines. The right border extends along the left edge of the sternum, from the fourth to the sixth rib. It is not so easily defined because of the peculiar wooden tone of the sternum when percussed.

The last statement not only corroborates the fact that the superficial area of marked and comparatively easily detected dulness is no indication of the size of the heart generally, but that neither does it indicate exactly all of that portion of the heart which is uncovered by lung; for a part of the right ventricle not overlapped by the margins of the lungs lies to the right of the left border of the sternum, and yet the percussion note over it (the lower third of the sternum), instead of being dull, has this clear, pulmonary resonant quality modified by the sounding-board vibrations of the bone, so that the dulness which the ventricle underneath would otherwise give is "overborne by the clearness of the pulmonary note" conducted back to the percussion point of transmission.

The *clinical value* of the superficial area of cardiac dulness is very much less than that of the deep or relative dulness, therefore, in estimating the size of the heart, because the extent of dulness varies with the position of the lung borders, which are themselves variable even in the healthy individual, due to the inspiratory and expiratory movements, and the conditions of age. As to the latter, the absolute dulness begins a little higher in children (third interspace), while in old age the extent of dulness is smaller, from senile inflation of the lungs, so that the upper boundary begins usually at the fifth rib, and the outer at the parasternal line.

Pathologically, although enlargement of the heart does cause some increase in the dimensions of the area of exposed dulness, by pressing the lung borders aside somewhat, shrinking of the left lung without such enlargement will manifest the same signs. Then, again, emphysematous encroachment upon the heart area will so diminish, or even obliterate, the dulness as to preclude entirely any indications of the cardiac size by this method.

The *deep or covered dulness*, however, is of decided importance as an index of the actual size of the heart, the limits of dulness corresponding with a much nearer exactitude to those of the organ itself, regardless of the position of the lung borders. The relative dulness is best demonstrated at the upper and outer limits of the heart area.

Thus it occupies a narrow zone of muffled resonance, the width of an interspace, beyond the left lung border—that is, beyond the upper and outer margins of the area of absolute dulness—being found in the *third interspace* inside the left parasternal line, and following a downward and outward course across the fourth interspace and fifth rib into the fifth interspace, at the apex, where it terminates. Hence, in the fourth and fifth interspaces, where the relatively dull zone is nearly vertical in direction, it lies between the parasternal and nipple lines. Within the apex point the deep dulness extends to the right in a straight line to join the absolute dulness in the parasternal line, and here immediately passes into the inseparable liver dulness as far as the right border of the heart, a little beyond the right sternal line. The right boundary of the covered dulness extends along the right edge of the sternum, from the fourth rib to the sternoxiphoid junction; it is detected with difficulty on account of the thickness of the lung tissue, and the fact that when the most forcible percussion strokes are applied close to or over the sternum the resonance set up by the vibration of the surrounding lung structure overshadows any

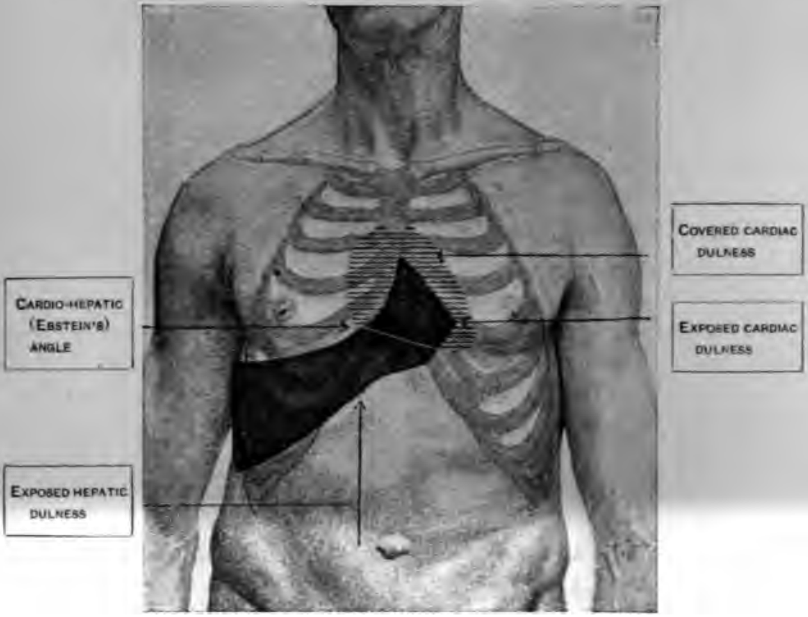


FIG. 45.—SHOWING THE EXPOSED AND COVERED DULNESS OF THE NORMAL HEART. (Butler.)

dulness elicited. And yet it should be borne in mind that the right auricle reaches as far as one and a half fingerbreadths to the right of the right border of the sternum. But in lateral enlargements of the heart this deeper dulness may be perceived here.

It will be seen, therefore, that the *total area of cardiac dulness* which can be mapped out by firm percussion, the superficial area bordered to the right, left, and above by the deep dulness, represents as nearly as possible the anatomic outline of the heart: laterally, from about 1 in. to the right of the right sternal margin in the fourth interspace to the nipple line in the fifth, and vertically, from the lower edge of the third rib, at the left of the sternum, to the upper edge of the sixth rib in the parasternal line, where it joins the hepatic dulness.

It should not be forgotten, also, that the heart is constantly, rhythmically changing its size with systole and diastole, so that the dermographic lines of delimitation will vary slightly with different examiners, or at different times with the same examiner if deeper or shallower breathing and corresponding changes in the extent of the lung borders are likewise factors.

Another point: Deep percussion over the sternum, above the level of the third rib, may develop a conoidal area of dulness rising above the cardiac area, but joined to it as the latter is to the hepatic below. This is due to the aorta and pulmonary artery, but principally the former. Aortic dulness, extending considerably beyond the sternal margins in the second interspace, would be suggestive of aortic dilation, either diffuse of the arch, as from fatty degeneration and weakening of the walls, or aneurismal.

Continued practise, concentrated attention, and careful judgment are always a *sine qua non* in estimating the separate or, more important, the entire areas of the heart dulness. In order to facilitate accuracy, a satisfactory technic or mode of procedure should be adopted and rigidly followed, once skill is thus acquired.

Technic of Cardiac Percussion.—We aim to determine the *entire area, limits,* and *shape* of the deep or covered heart dulness with as much accuracy as possible. The pleximeter finger should be applied firmly and evenly in the interspaces, and the plexor strokes should be given rather forcibly. With an anatomic conception of the usual topographic and relational extent and limits of the heart, the examiner endeavors to approach these in lines perpendicular to the supposed outline, beginning always over fully resonant lung tissue, and noting instantly with the ear the slightest elevation of pitch and change in quality and intensity, as well as any increased sense of resistance. Wherever the relative dulness is first noticed a mark may be made with a greased crayon, a line connecting the marks representing the probable outline of the heart.

While, in approaching the area of cardiac dulness, it is proper, if not almost natural, to place the pleximeter finger parallel to the supposed border of the heart, a word of caution is needed in estimating the lateral extent of the area. Here, of course, the finger must be placed across the ribs, the percussion of which interferes with the true note of the underlying structures; but by percussing the finger tips applied in the interspaces only, either at right angles or parallel with the ribs, confusing and misleading bone sounds may be eliminated.

From the position and relations, and from the formation of the heart, it must be apparent that although we can percuss out nearly its entire limits, for practical purposes this is not necessary; in most cases it will suffice to ascertain merely the extent of cardiac dulness along two lines—viz., a vertical and a transverse one. For the first one we use the parasternal line (left); that is, about 1¼ in. to the left of the sternal margin. Percussing strongly, we begin in the second interspace in this line. We find here, in the normal condition, the clear, loud, low-pitched lung resonance to the upper border of the third rib. In the third interspace there is a slight rise in pitch, diminution in intensity and duration, and dulling of quality of the sound. Below the lower border of the fourth rib the note has become quite dull and higher in pitch; in the fifth interspace it is flat, short, empty, or toneless. The rise in pitch by these successive gradations, as in playing the notes of a musical scale, I have been in the habit of demonstrating by applying simultaneously the first, second, and third pleximeter fingers (from below upward) in the second, third, and fourth interspaces, and percussing them in rapid order. Below the sixth rib the absolute dulness becomes again relative, and then clear because of gastric and intestinal tympany.

Similarly, in marking out the lateral or transverse boundaries we percuss from without inward until a rise in pitch signifies that we are over airless (cardiac) structure. On the left side we may delimit the curving, oblique heart border by percussing inward in each interspace, beginning in the first or second, until dulness is reached in the slightest degree; in the same way the third, fourth, and fifth interspaces are percussed from lung to heart, always beginning far enough out so as to obtain the pure resonance or, as it were, standard or keynote, the first dulling or acoustic deviation from which we are to perceive. "In this way the true apex of the heart is found, a point of importance when auscultation is followed, and which is to be distinguished from the precordial pulsation in other positions, often erroneously called the apex-beat" (Gibson and Russell). To define the right boundary of the organ, one proceeds in a corresponding manner, from the right midclavicular or parasternal line toward the sternum, first determining the upper boundary of deep or covered liver dulness, however, which is usually in the fifth interspace (rarely the fourth), and then percussing successively in the second, third, and fourth interspaces until relative dulness is elicited with forcible strokes.

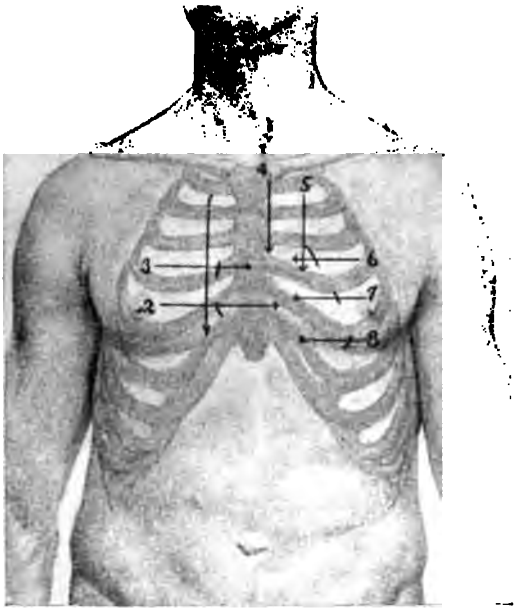


Fig. 46.—Showing the Principal Lines (Numbered in Order of Performance) along which Percussion should be Conducted to Ascertain the Area of Cardiac Dulness, both Covered and Exposed. Compare with Fig. 45. (Butler.)

That there is more technical difficulty in outlining the right edge of the heart by percussion is explicable from the fact of the "depth of lung between the parietes and the part of the heart to the right of the sternum" being considerable, and because "the difference in sound when we pass from lung to lung with underlying heart is correspondingly slight."

While the delimitation of the lower border of the heart is of much less importance, and rather difficult *per se*, a few practical points may be heeded which usually avail. In the first place, a line drawn joining the inferior limits of the markings of the lateral boundaries represents this border fairly accurately. Furthermore, this trend may be indicated by a finger's breadth of tympanoresonance just below and a little distance inside the apex, between the cardiac and hepatic dulnesses. Even within the parasternal line, however, where the dulness joining liver and heart is almost, if not quite, uniform on superficial examination, the transition may, nevertheless, be detected very frequently by perceiving a difference in the character of the dulness and the sensation of resistance. According to Gibson ("Diseases of the Heart and Aorta," p. 137), the best method of ascertaining this lower boundary of the heart is to "map out the entire areas occupied by the two organs, when a line drawn from the angle where the margins—ascertained by the deep dulness of the two viscera—meet, to the right of the sternum, is to be drawn to the point where the left end of the liver and the lower margin of the heart are found to be in contact."

Exceptions.—In women, on account of the size and sensitiveness of the mammæ, it may be impossible to determine the size of the heart by percussion. Recourse then may be had to an approximation of the size by locating the apex-beat by palpation. If normal, the latter is found ½ or 1 in. within the midclavicular (not nipple) line in the fifth interspace.

Again, in young children, owing to the thinness and elasticity of the chest walls, percussion must be practised with extreme lightness of stroke, so as to avoid setting into vibration the adjacent clear lung and stomach sounds to the confusion of the cardiac dulness. Also, it must not be forgotten that in children the area of superficial or exposed dulness is relatively greater at the upper and outer borders, extending to the third intercostal space.

Other methods, such as Sansom's pleximetric percussion, repercussion, palpatory and auscultatory percussion, have certain auxiliary and precise values, but are ordinarily impracticable.

ABNORMAL AREAS OF CARDIAC DULNESS

The area of cardiac dulness may be *increased, diminished, abolished*, or *dislocated*.

(1) Increase of Heart Dulness. (*a*) HYPERTROPHY AND DILATION OF THE HEART.—Enlargement, due to the usual combination of thickened and overstretched walls of the heart (hypertrophic dilation), is inferred by the following physical signs of topographic percussion: If the area of dulness (deep) is increased in all directions, but especially more transversely than upward, in the fourth and fifth interspaces, the dilation and hypertrophy probably affect both ventricles, the extreme left border of the dulness being found a trifle outside the apex-beat. When hypertrophy predominates, a powerful apex-beat and cardiac shock are palpable, while when dilation supervenes, the shock may be absent, and the apex-beat diffuse and weak. The dulness may be detected 1 in. beyond the left midclavicular line, also to the right of the sternum as high as the fourth costal cartilage. To the left of the sternum, as far as the parasternal line, the dulness may be more marked than normal in the third interspace. Over the sternum, below this level, the resonance is likely to be muffled slightly.

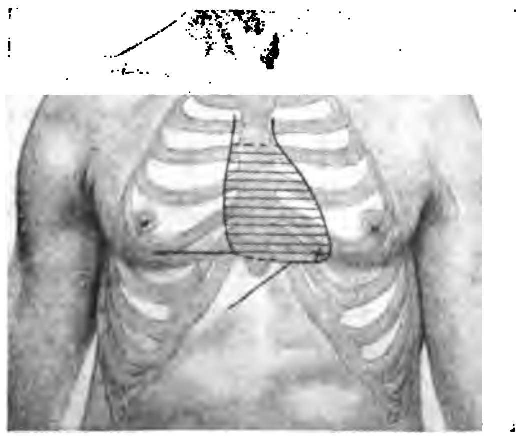


FIG. 47.—NORMAL AREA OF ENTIRE CARDIAC DULNESS. The dotted lines above and below represent the borders which are difficult to delimit, but the apex-beat and the finding of the solid-lined portions of the borders enable a satisfactory determination of the size and shape of the heart. (Butler.)

Increase of dulness to the left and slightly downward indicates enlargement of the left ventricle. If the area of dulness over the apex is pointed, and the apex-beat is strongly palpable, hypertrophy predominates. If, on the other hand, the extension of dulness to the left has a rounded apical outline, with a wavy, diffuse apical impulse felt propagated to the right (within the apex region), dilation is predominant. In the extreme cases of hypertrophic dilation of the left ventricle, due to the prolonged effect of insufficiency of the aortic valve, the left border of deep or relative dulness may reach the mid-

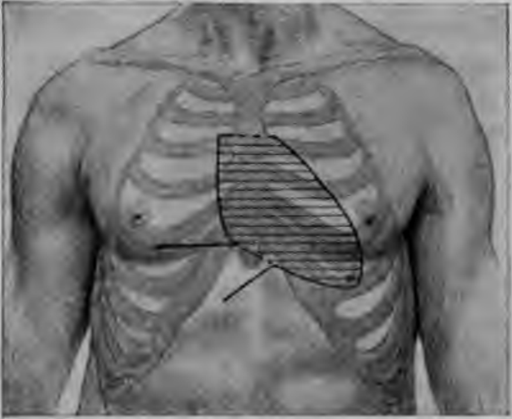


FIG. 48.—DULNESS IN HYPERTROPHY OF THE LEFT VENTRICLE. Apex-beat heaving and carried down and to the left, perhaps outside of the apex outline. Apex pointed. (Butler.)

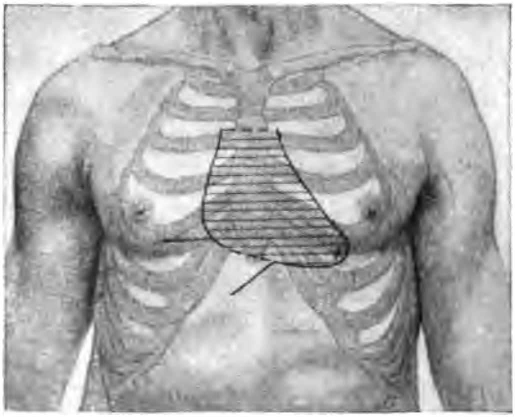


FIG. 49.—DULNESS IN HYPERTROPHY AND DILATION OF THE RIGHT HEART. Note apex-beat moved to the left, and dulness increased to the right of the sternum. (Butler.)

axillary line in the sixth or seventh interspace, and the absolute dulness as far as the anterior axillary line nearly.

Increase of the dulness to the right, especially if noted in the third and fourth interspaces, points to enlargement of the right auricle (which is quite dilatable in direct and indirect obstructions to the flow of blood), and of the right ventricle. But in cases where the latter is much dilated, there is simultaneous extension of the left border of dulness, owing to the more horizontal position which the heart assumes, which displaces the apex somewhat to the left. The sternal resonance is modified.

With marked hypertrophy of the left ventricle, the general shape of the cardiac dulness preserves a triangular outline, while with dilation of the right or of both ventricles, a quadrilateral outline is more commonly detected.

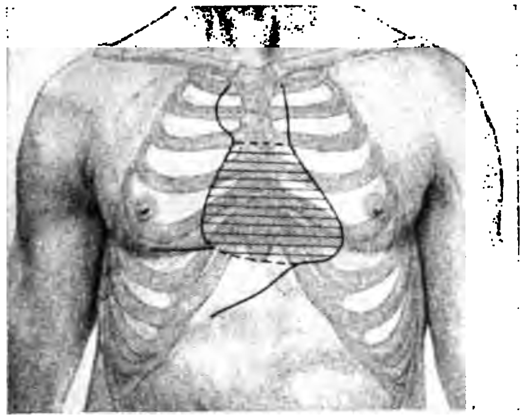


FIG. 50.—SHOWING THE DULNESS DUE TO DILATION AND HYPERTROPHY OF BOTH VENTRICLES. Apex rounded and apex-beat diffused. Compare with Figs. 48 and 49. Shows also (first and second right interspaces) the dulness of aortic aneurism. (Butler.)

Circumscribed dulness in the second left interspace, close to the sternum, is quite constant in mitral stenosis.

(*b*) PERICARDIAL EFFUSION.—*Simultaneous extension of the area of cardiac dulness toward both sides and upward* denotes a liquid effusion into the pericardial sac. If the quantity of fluid is large, the area of dulness takes the form of the distended sac, and thus is irregularly triangular or pyramidal (sometimes almost equilateral) in outline, the base being downward, where the enlargement begins and shows the most. The limits of dulness to the right and left here depend upon the size of the effusion; if very abundant, the base of the triangle may extend from the right parasternal line in the fifth interspace to the left anterior axillary line almost. At the same time the apex of the dull area may reach to the second interspace, where the converging outlines of dulness meet from below, to form a rounded obtuse angle. The distention of the lower portion of the pericardial sac is recognized by the fact that the apex-beat, or on auscultation the point of greatest intensity of the first sound, is found to be well inside the limits of dulness. A less considerable quantity of exudation gives rise to a proportionately smaller area of dulness, extending probably but a trifle beyond the nipple line, within which the apex-beat may be felt.

It is distinctive that in large pericardial effusions the area of dulness is altered by changing the position of the patient, in the sitting posture being larger than in the recumbent; the sternal resonance may then be abolished. Another feature is the absence of any distinction between the deep or covered and the superficial or exposed dulness, the overlapping lung being pushed aside and leaving the uniform dulness of the distended sac. Again, for the same reason, it is characteristic that, in approaching the dull area, a narrow zone of tympanitic resonance is percussed just before an abrupt transition to the effusion flatness. When the fluid happens to be the mobile transudation of hydropericardium, changes in the clearness or dulness of the borders, or of the extent of dulness, on changing the patient's position to the back or either side, are still more evident.

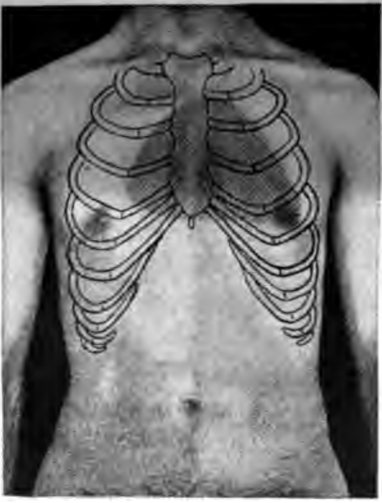


Fig. 51.—Absolute Dulness in Case of Acute Pericarditis. (Babcock.)

(*c*) Apparent Increase of the Cardiac Dulness and Size of the Heart.—In the *first* place, this may be due to retraction of the lung over a normal heart, the determination of the complete outline of which will show that the exposed area of cardiac dulness only is affected; the absence of mobility of the border of the lung confirms the fact of the apparent increase of heart dulness. *Secondly,* this may occur also as a result of a pushing forward of the heart by a tumor or aneurism in the posterior mediastinum. In the *third place*, apparent increase of cardiac dulness may be observed where the adjacent lung is consolidated or the pleura thickened. But by the method of percussion alone it may be impossible to determine where heart dulness begins and pulmonary or pleural dulness ends; the position of the apex-beat, the character of the vocal fremitus and breath sounds must be solicited as aids in the diagnosis.

(*d*) It remains to note actual INCREASE OF THE PRECORDIAL DULNESS UPWARD, because of disease of the aorta especially. Extension of dulness above the normal cardiac area, and to the right of the sternum, usually means *aneurism* of the ascending portion of the aortic arch; over the manubrium, of the transverse portion; to the left of the sternum, in the first and second interspaces, of the descending portion at its beginning. Aneurismal dulness is thus commonly a smaller, rounded area of dulness, superposed and connected with the larger, triangular area of cardiac dulness.

(2) **Diminished and Absent Cardiac Dulness.**—As the area of absolute or exposed cardiac dulness depends upon the extent of the overlapping lung borders, and as the deep, relative, or covered dulness depends upon the thickness of the same as well, it is evident that any diminution of these areas (but especially of the superficial area) must mean some encroachment upon the boundaries of the heart by air-containing tissue. This condition is most typically illustrated in cases of *emphysema* of the lungs. In very marked instances of the latter the cardiac dulness may be totally absent. In moderate cases the heart outline may be percussed by the heaviest strokes. As the heart in advanced cases of emphysema is frequently enlarged (right ventricle), there is often difficulty in detecting it by percussion because of the interposed enlarged lung. So that a normal-sized area of cardiac dulness, with the existence of emphysema, would really indicate a considerable enlargement of the heart.

The heart dulness is absent, also, owing to the presence of air in the pericardium—*pneumopericardium*—from injury, as stab wounds; there is then a tympanitic resonance heard in percussing over the heart. Similarly, the juxtaposition of a *left pneumothorax*, or of a gaseous distention of the stomach, may prove deceptive in causing a loud, deep resonance or tympany in the præcordial area.

(3) **Displaced Cardiac Area of Dulness.**—Whether the heart dulness, in cases of its dislocation, can be elicited or not depends upon the cause. If within the reach of the percussion strokes at all, its

size and outlines will correspond with those of the heart itself pretty nearly. The causes of displacement have been given under inspection and palpation, in connection with displacements of the apex-beat; indeed, when palpable, the position of the apex-beat aids materially in locating the heart dulness. Even then it may be impossible to delimit the cardiac dulness throughout, when, as often happens, the cause of the displacement as a pleuritic exudation itself gives dulness on percussion.

The dislocated area of dulness of a dislocated heart may thus be normal in size; it may be smaller than normal, as when the heart is drawn under and away from the chest wall by pleural adhesions and shrinking of the lung, or it may be increased because of a larger parietal contact with the chest wall when the heart is pushed up against it by a high position of the diaphragm. Moderate displacement of the heart to the right, under the sternum, as from emphysema, is designated as *mesocardia*. Extreme displacement to the right, as in very large left-sided pleural effusions, or in transposition of the viscera (*situs inversus*), is known as *dextrocardia*.

CHAPTER XII

AUSCULTATION

Auscultation may be considered the diagnostic climax in the physical examination of the heart; not necessarily a method of independent value and reliability, but one which affords the most positive, precise, and complete data, without which those obtained by inspection, palpation, and percussion would, combined, fall far short of indicating the physical condition of the heart and its valves. Indeed, the perfection or abnormality of the cardiac mechanism may, in most cases, be estimated fairly well from the character of the cyclic sounds alone. Obviously, to determine and differentiate as fully and accurately as possible the significance of variations of the natural and adventitious cardiac sounds, auscultation should always follow the other methods of exploration. No method is sufficient without the others; the data obtainable by all four methods must be collected, collated, and analytically considered before any diagnostic conclusions are properly deducible.

The ***object*** of the auscultator is to find out the character of the ***heart sounds***, and then to detect and study any *adventitious sounds* (***murmurs***) that may be heard.

METHOD OF EXAMINATION

Immediate auscultation may at times be employed by an experienced and skilful examiner, to ascertain in a general way, as in emergency, the condition of the heart sounds or the presence of a murmur. But no accurate work can be done without the use of a stethoscope. My preference is exclusively for the binaural, with a small chest piece, so that it may be placed evenly in the interspaces, and so that the precise points of origin of the particular physical signs may be sharply localized. Whatever form of stethoscope is adopted by the physician, whether the single, ordinary binaural, Bowles', or other pattern, let him use that which continually gives the best and most satisfactory results to himself, no matter how favorable the recommendations of other instruments by other men. It may be added, however, that by immediate auscultation, especially in cases of hypertrophied and dilated heart, one may occasionally better determine the time during the cardiac cycle when the normal and abnormal sounds occur by noting the time when the impulse is felt by the tactile sense of the ear. Usually the practise is to palpate the carotid artery with the finger, and thus compare its pulsation with the heart sounds or murmurs.

The patient should be at rest in body and serene in mind, preferably in a comfortable sitting posture, so as to bring the heart in close contact with the chest wall. Sometimes it may be necessary to have him make a little exertion when the sounds are very weak. The resulting increased activity of the heart and intensity of the sounds may be obtained by having the patient sit up in bed, or turn over several times, or, if out of bed, bend the body backward and forward a few times while standing, or step off a few paces briskly.

THE NORMAL HEART

NORMAL HEART SOUNDS

When the stethoscope is applied over the body of the heart, in the third and fourth interspaces, within and in the left parasternal line, we hear sounds and silences or pauses rhythmically alternating. Indeed, they may be distinctly audible all over the precordium, and for some distance beyond it on both sides of the chest; faintly, even posteriorly, in certain individuals with thin thoracic walls. The predominant sound is found to be synchronous with the apex-beat and carotid pulse as palpated with the finger, and hence is called the *systolic* or *first sound*, because it coincides with these phenomena of the systole or ventricular contraction of the heart. An extremely short pause follows, which is in turn followed by a different sound, the *second*, or *diastolic*. This sound occurs at the beginning of a longer pause, corresponding to the diastole. These two sounds are often traditionally represented by the respective monosyllables, *lub-dub*. Thus the first sound and short pause, second sound and long pause constitute a complete cardiac cycle. This rhythm of sounds and silences is not apparent in infants, where the former are alike in character and the latter in duration, similar to the uniform ticking of a watch. In the adult, therefore, how do we distinguish between these two sounds in themselves? The answer is: By their attributes.

First Sound.—The *quality* of the systolic sound is a dull booming "lub." The *intensity* is comparatively marked. The *pitch* is relatively low. The *duration* is long.

Second Sound.—The *quality* of this sound is sharp, snapping, or clicking. Its *intensity* is less loud than that of the first sound. The *pitch* is distinctly higher than the other. The *duration* is shorter. In other words, the first sound is heavier, louder, lower, and longer than the second; or, conversely, the second sound is snappier, softer, higher, and shorter.

Although the first and second heart sounds may be heard over the whole precordium, their accent varies at different points, while the rhythm remains the same. Thus, since the first sound corresponds with the ventricular systole, its accentuation is heard at that point where the ventricular conduction of sound is best heard, namely, at or near the apex. On the other hand, the second sound is accentuated at the base of the heart, corresponding as it does with the general location of the aortic and pulmonary valves, whose simultaneous closure at the commencement of diastole is the physiologic explanation of this sound.

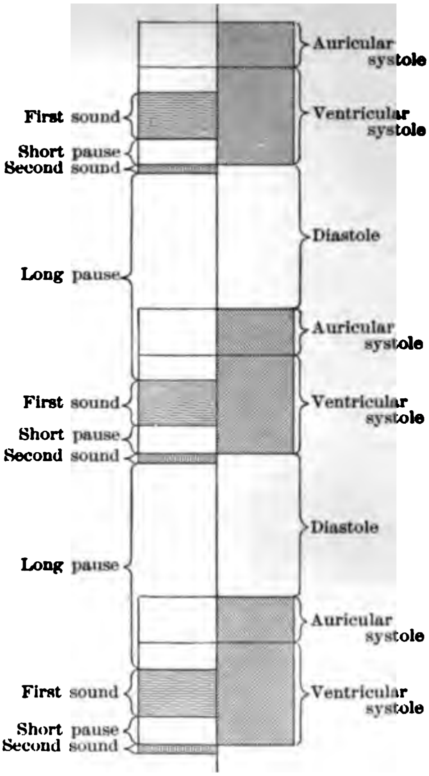


FIG. 52.—NORMAL CARDIAC CYCLE. Phases of cycle to the right of line; sounds to the left. (Babcock.)

Causes of the Heart Sounds.—The *first sound* is produced by the synchronus closure of the mitral and tricuspid valves, and the synchronous contractions of the right and left ventricles. Probably the sudden rush of blood due to the filling of the beginnings of the pulmonary artery and aorta (the *coni arteriosi*), and the impact of the apex against the chest wall, are contributory factors also.

The *second sound* is undoubtedly the result of the sharp closure, synchronously, of the pulmonary and aortic valves. Thus it will be noticed that both sounds are caused practically by valvular action, although, as will be indicated later, the character of the first sound may be altered considerably in affections influencing the vigor of the muscular contractions primarily, and thus, in a measure, the force of closure of the auriculoventricular valves secondarily.

The Cardiac Cycle.—The mode of production of the two heart sounds will be understood better, perhaps, by rehearsing the physiologic movement of the blood through the heart, and then simply summarizing the actual cardiac movements themselves as they blend in normal rhythm. Concerning the former, I may quote with advantage the clear and graphic statements of Vierordt: "The blood flows from the body through the *cavæ* into the right auricle, from whence, during the ventricular diastole, it passes through the right auriculoventricular opening, the tricuspid valve, into the right ventricle, being urged forward toward the end of the diastole by the weak muscular contraction of the right auricle. The systole which immediately follows drives the blood out of the ventricle, the tricuspid valve being at the same time closed, through the open pulmonary semilunar valve into the pulmonary artery. The blood, prevented from flowing back into the ventricle during the diastole, which immediately follows, by the closure of the pulmonary semilunar valve, passes through the lungs, and from them flows into the left auricle, whence, by the diastole of the ventricle, it flows through the left auriculoventricular opening, the mitral valve, into the left ventricle, whither it is again assisted at the end of the diastole by the contraction of the auricle. The left ventricle discharges its contents during the systole (the mitral valve being closed) into the commencement of the aorta, through the open aortic semilunar valve, whence it is prevented from returning to the ventricle when the pressure from the ventricle ceases and the diastole begins, by the closure of the aortic semilunar valve. The blood then flows from the *conus aortæ* into the body."

In the second place, however, it should be remembered that the blood enters the aorta and pulmonary artery at the same time by the synchronous contractions of the two ventricles. With the completion of this systolic unison, relaxation of the ventricles ensues, and at once the recoil of the arterial walls forces the columns of blood back against the semilunar valves, which close with the snap of sudden tension thus at the commencement of the ventricular diastole. Hence, four sounds are created, one at each valve orifice, normally audible as two because of the simultaneous closure of each homologous pair.

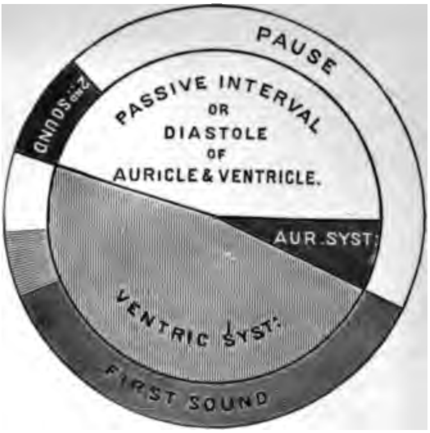


Fig. 53. — Diagrammatic Representation of the Movements and Sounds of the Heart. (After Sharpey.) This diagram shows merely the general relations of the several events, and does not represent exact measurements. In a heart beating 72 per minute, Foster estimates each entire cardiac cycle as occupying about 0.8 second, of which 0.3 second represents the duration of the systole of the ventricles, 0.4 second the diastole of both auricles and ventricles, or the "passive interval," and 0.1 second the systole of the auricles. (Le Fevre.)

In connection with the first or ventricular systolic sound, Babcock writes as follows: "Physiology teaches us that the duration of this phase of the cardiac cycle is $\frac{35}{100}$ of a second, subdivided as follows: During the first tenth of a second the ventricle is initiating its contraction and is silent; during the following $\frac{15}{100}$ of a second its contraction reaches its maximum energy, the auriculoventricular valves close, and the first heart sound is heard; the final tenth of the second, during which the ventricle still remains contracted, is again a period of silence, and terminates the phase of ventricular systole."

To *summarize*: The *rhythm* or *time* is the basis of distinction between the first and second cardiac sounds, apart from their normal characteristic attributes, the first or systolic sound being that which is heard at the time of the normal cardiac impulse or carotid pulse, or just before the radial pulse, the second sound being the one which follows the impulse. Also, the sound which follows the long silence is the first sound; that which precedes the long and follows the short silence is the diastolic or second sound. The systolic sound is partly (mainly) valvular and partly muscular; the other purely valvular.

From the preceding it is apparent that the valves are very essential in the production of the heart sounds, and therefore that any alterations of these sounds must mean some imperfection in the function or structure of one or more of these valves or their orifices. But the heart is double, and the problem in physical diagnosis is to determine whether the right or the left auriculoventricular valve is at fault in causing any abnormality of the first or systolic sound, which is synchronously produced by both, and whether the right or left semilunar valve is diseased when the separately, yet synchronously, produced second sounds are altered. Furthermore, as the adventitious heart sounds known as murmurs occur in connection with affections of the valves, it is of first importance to know which of the four valves is the seat of the lesion.

VALVE AREAS

Naturally, one would auscult at that point which is nearest to any particular valve and orifice, but experience has shown that the clinical areas of differentiation do not correspond with the anatomic positions of the valves. Thus, a superficial area of half an inch square will include a portion of all four sets of valves, so that stethoscopic examination here fails to detect the valve affected. We cannot tell a right first or second sound from a left first or second sound, or at which orifice a murmur is developed.

The respective *valve areas* selected for auscultating the heart represent the points to which the vibrations from the corresponding valves are best conducted, and exhibit, therefore, the greatest intensity. We have, then, the mitral area, the tricuspid area, the aortic area, and the pulmonary area. The mitral and aortic valve sounds are most audible at a greater distance from their anatomic positions of origin than are the tricuspid and pulmonary because of their deep location, the first being behind the overlapping lung, the second behind the pulmonary artery. On the other hand, the tricuspid and pulmonary valves are comparatively near the surface of the chest wall, and consequently are productive of sounds which are distinctly audible near their anatomic origins.

Areas of Maximum Intensity.—The locations of greatest intensity of the first, systolic, or ventricular sound—near the apex—and the second, diastolic, or arterial sound—at the base of the heart—have already been indicated.

(1) THE MITRAL AREA.—The sound produced by the closure of the mitral valve (at systole) is best heard at the *apex,* within an area about 1 in. in diameter. It is more audible here than over the precise situation of the valve, because, in its deep origin, it is conducted by the walls of the left ventricle to that point where the latter lies most closely to the thoracic wall, namely, the impinging tip.

(2) The Aortic Area.—The *second right intercostal space* near the border of the sternum is the point of maximum loudness of the sound of closure of the aortic valve leaflets. This is because the sound is propagated along the course of the blood current to that portion of the ascending, tense-walled aorta which is nearest to the surface of the chest at the second right cartilage.

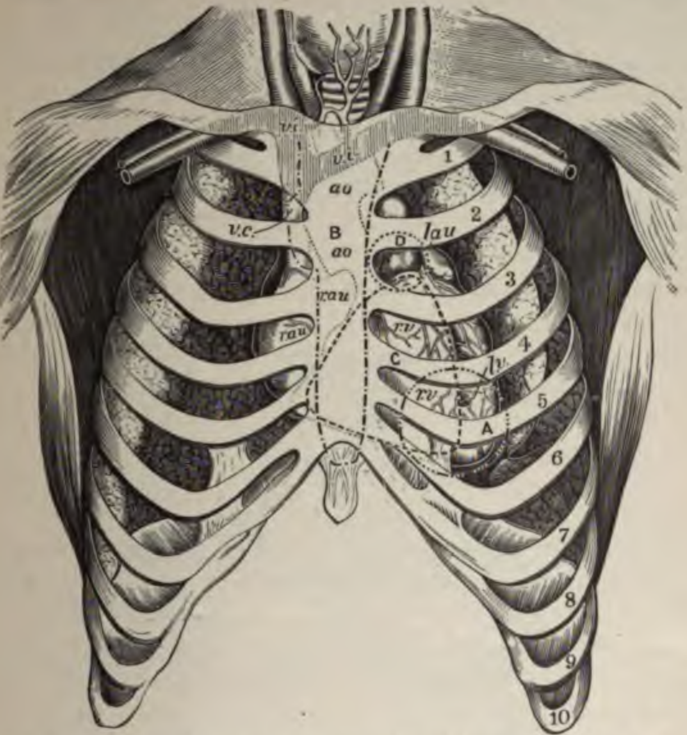


Fig. 54.—Areas of the Cardiac Murmurs. (After Gairdner and Luschka.) The outlines of organs, which are partially invisible in the dissection, are indicated by very fine dotted lines; while the areas of propagation of valvular murmurs, as described in the text, have been roughly marked by additional much coarser and more visible dotted lines—the character of the dots being different in each of the four areas. A capital letter marks each area, viz.: *A*, the circle of mitral murmurs corresponding with the left apex; *B*, the irregular space indicating the ordinary limits of diffusion of aortic murmurs, corresponding mainly with the whole sternum, and extending into the neck along the course of the arteries; *C*, the broad and somewhat diffused area occupied by tricuspid murmurs, and corresponding generally with the right ventricle; *D*, the circumscribed circular area over which pulmonic murmurs are commonly heard loudest. Reference letters: *r. au.*, right auricle; *a. o.*, arch of aorta; *v. i.*, the two innominate veins; *v. c.*, vena cava descendens; *p.*, pulmonary artery; *l. au.*, left auricle; *l. v.*, left ventricle; *r. v.*, right ventricle. (Finlayson.)

(3) THE TRICUSPID AREA.—The point of election in auscultating the tricuspid element of the first sound is at the *lower part of the sternum*, especially near the left border opposite the fourth and fifth interspaces. It is in this region that the right ventricle, uncovered by lung, is in close contact with the chest wall.

(4) THE PULMONIC AREA.—The closure of the pulmonary valve is heard best at the *second left interspace* near the sternal border, or at the sternal end of the third left costal cartilage, directly over the valve itself. This is the only one of the four areas for auscultation, therefore, which coincides practically with the anatomic position of the valve.

The points of election just given are used in localizing the seat of production of organic valvular murmurs even more than for the purpose of differentiating the separate valve sounds themselves, although the characters and changes of the latter should be studied more frequently and carefully than is commonly done.

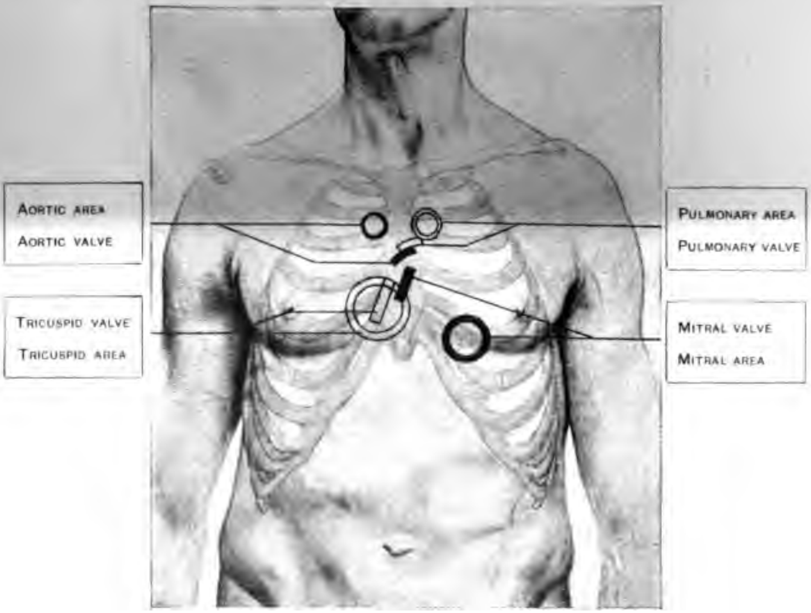


FIG. 55. SHOWING THE POSITIONS OF THE VALVES OF THE HEART AND THE AREAS OF THEIR GREATEST AUDIBILITY. Solid circles and blocks = deep valves (aortic and mitral). Light circles and blocks = superficial valves (pulmonary and tricuspid). (Butler.)

Vierordt summarizes the physiologic events producing the first and second sounds as heard at the four valve areas in the following manner:

"Apex of the heart (*mitral orifice*):

"First sound: Closure of the mitral valves and ventricular contraction.

"Second sound: Prolonged aortic second sound (closure of aortic valve).

"Under the sternum (*tricuspid orifice*):

"First sound: Closure of the tricuspid valves and ventricular contraction.

"Second sound: Prolonged pulmonary second sound.

"Second intercostal space, right or left (aorta, pulmonary artery):

"First sound: Sudden filling of the beginning of the aorta, of the pulmonary artery, and continuation of the first ventricular sound.

"Second sound: Closure of the semilunar valves of the aorta, or of the pulmonary artery."

PHYSIOLOGIC VARIATIONS OF THE HEART SOUNDS

Within normal limits, the heart sounds may vary in *strength* or *loudness*. This is true usually of both sounds together; that is, they are both relatively increased or diminished in intensity. The condition may be temporary, as the increase of intensity due to a full, stimulating diet, physical exertion, or mental excitement, or more or less persistent, as the increase from thinness of the fleshy coverings of the chest, as well as the flexibility and delicacy of the bony thorax in children and certain delicate adults. On the other hand, in those with thick, obese chest walls, and especially women with large, fat mammary glands, both sounds—the first, usually, more than the second, however—are relatively, yet normally, weakened. The heart sounds are also less distinctly heard in an individual lying down on the back or reclining than when in the upright position, owing to the organ's swinging back from the precordial region.

Thus it is apparent that the loudness of the cardiac sounds depends upon the nature and thickness of the media through which they have to pass, the degree of blood pressure within the heart and arteries, and the distance of the vibrating valve orifices and ventricular muscle from the front surface of the chest.

The *individual valve sounds* vary also, intrinsically, and according to the factors of *age, temperament, vigor of health,* and *character of* *nervous system, occupation*, etc. Not only intensity, but normal quality, pitch, duration, and rhythmic variations are frequently observed.

In childhood, the valvular element of the *first sound* predominates over the muscular, and has a more ringing, higher-pitched, shorter character than in the adult. In the latter, the first sound has a duller, more booming quality, of lower pitch and longer duration, although in certain thin, nervous people it may partake of the type of early life. In the very vigorous and robust the first sound is often of a loud, prolonged, rumbling nature, while in the fat and indolent it is distant, indistinct, and short. The sound as heard in the tricuspid area is of about equal character and intensity as that of the mitral area, although at every age the latter, or sound produced by the left ventricle, is more likely to be a little heavier than the other, if there is any difference at all.

The *second sound* is, of course, louder at the base than at the apex. Its component elements, however, differ in relative intensity. In childhood the pulmonic sound is the stronger of the two, in middle life they are about equal in loudness, while in advanced age the aortic sound predominates over the pulmonic, probably because of the senile degenerative changes. As to *pitch*, in a series of nearly 500 healthy individuals I have found that in about eighty per cent the aortic sound was slightly, but distinctly, higher than the pulmonic, the ages ranging from twenty-two to fifty years principally. Hence, the term *accentuation*, which is generally applied to variations of the intensity of the sounds, should not be used without careful discrimination, and the proper designation of the attribute which may be qualified. For if the aortic sound is referred to as accentuated, for example, the diagnostic interpretation and value of the physical sign will depend more or less considerably upon whether intensity or pitch is meant. In regard to *pitch*, the designations *elevation* or *depression* may be used here, just as they appropriately describe variations of this attribute in other connections.

The *rhythm* of the first sound may be physiologically disturbed in a so-called *doubling*, whereby the sound is divided, but yet without any interval such as exists between the first and second sounds. It may be likened to the pronouncing of the syllables "turrupp" or "trupp." This phenomenon may occur in health at the end of a deep inspiration and holding the breath, although this is much more common as a cause of doubling of the normal second sound. In fact, doubled first sound is rarely physiologic, and is more apparent than real, the splitting of the sound being probably due to a transient peculiarity whereby the "maximum intensity of the left first sound does not coincide with the maximum intensity of the right first sound." Or possibly the doubling may be due to temporary excitement of the heart, in which one hears the sudden shock communicated to the ventricular contents just before the occurrence of the systole.

Reduplication or division of the *second sound*, while also occurring normally sometimes, is usually pathologic. It is likewise heard in some individuals at the end of a deep inspiration, the intracardiovascular pressures being altered for the moment, so that the pulmonary and aortic valves fail to close simultaneously.

PATHOLOGIC MODIFICATIONS OF THE HEART SOUNDS

Changes in the character of the heart sounds, individually or collectively, may be observed in diseased conditions in connection with one or more of the attributes, although the most important alterations pertain to the intensity, pitch, and rhythm of certain sounds as regards diagnostic and, sequentially, prognostic and therapeutic significance and trend.

Quality.—The *first sound* may become sharper and more snapping, like the second sound, and at the same time shorter and higher pitched, although the actual volume (loudness) of sound is diminished. This flapping quality of the first sound indicates weakness or thinness, or both, of the ventricular walls, as met with in cases of marked, chronic debility from *anemia*, chronic *tuberculosis*, and the like, and in those *phlegmatic* and *neurotic temperaments* which are often hereditary and sometimes acquired in certain walks of life. This modification of the first sound is not infrequently noticed in cases of obstruction of the mitral orifice, with consequent atrophy and weakness of the left ventricle.

The *second sound* may have a kind of clanging, flopping, or booming quality, also lower in pitch. I have found this to be most distinct in the second right interspace, in very old persons giving evidence of loss of aortic elasticity from fatty degeneration of the walls of the arch, with dilation of the aorta; also in cases of weak ventricle, the systemic arteries being normal.

Metallic quality of both heart sounds indicates usually a large, smooth-walled pulmonary cavity near the heart, a *pneumopericardium* or a *pneumothorax*, or an *inflated stomach.*

Intensity.—Changes in intensity or loudness may affect both sounds equally, or they may affect but one sound, either by way of increase—*accentuation*—or diminution.

(1) Increased Intensity of Both Heart Sounds.—Essentially this will occur in all conditions in which the blood pressure of the pulmonary or systemic circulations is increased. Overaction of the heart, therefore, from whatever cause, transient or permanent, usually accounts for abnormally increased intensity of the heart sounds, provided that certain adjacent changes may be eliminated. These may be pleural or pulmonary conditions, causing the heart to be uncovered more than normal; *consolidation of the overlapping lung*, which facilitates and exaggerates the conduction of the heart sounds; juxtaposed *cavities in the lung* and *pneumothorax*; and, at times, *adherent pericardium*.

Intensification of heart sounds from overaction is witnessed in cases of *cardiac hypertrophy*, nervous excitement and palpitation, exophthalmic goiter or Basedow's disease, and in certain febrile and anemic states.

(2) Diminished Intensity of Heart Sounds.—Weakening of the heart sounds may be due to causes in the heart itself, or to the muffling effect of interposed structures. Of the former should be mentioned the weakened heart muscle from the various causes of degeneration, often accompanied with dilation, such as the *toxemias of prolonged fevers, septic conditions, carbonic-acid and other poisoning, myocarditis, fatty degeneration, cardiac weakness from general exhaustion*, as in shock, hemorrhage, and malignant disease, and the *loss of compensation in a hypertrophied heart with valvular lesions.* Closely related to these affections is the central or peripheral paralysis of the pneumogastric.

Under the second category may be put the conditions which displace the heart or separate the heart from the chest wall pathologically. *Emphysema of the lung*, the enlarged margin of which masks the cardiac sounds by its greater overlapping, *pericardial and pleuritic effusions*, belong here. Finally, the heart sounds may be weakened relatively by the interfering effect of loud râles in adjacent parts of the lungs.

(3) Modified Individual Sounds. *Increased Intensity or Accentuation of the First (Systolic) Sound.*—As in the case of the two heart sounds considered together, conditions which raise or depress the power of the ventricles, or the pressure of the systemic or pulmonary vascular function, will correspondingly and respectively in-

tensify or lessen the first sound. Accentuation of this sound is commonly of two varieties: either the increased loudness is associated with a dull booming, grave, long character, or it is of a flapping, short and sharp character. The first variety is found typically in cases of marked *hypertrophy of the ventricle* (*left*, especially) or ventricles, and this is usually followed by an abnormally loud second sound. The peculiar quality of accentuation of this sound of ventricular hypertrophy may be due to a degree of exaggeration of the muscular over the valvular elements.

The second variety of accentuated systolic sound is often due to moderate dilation and weakness of previously hypertrophied heart walls. The valvular component predominates here, and the high-pitched sound has the snappy character of the "irritability of weakness." This modification of intensity is noted also in some athletes who, after a season of severe efforts in competitive sports, cease or relax careful habits of training and physical culture; also in many of those persons who are conveniently classed as "neurotic," and at the onset of acute febrile diseases.

A phenomenally intense, slapping character of the first sound is considered almost diagnostic of *mitral stenosis* by some experienced clinicians. Whether or not the accentuation of the first sound is due to increased action or intraventricular pressure on the right or left side will depend mainly upon stethoscopic localization over the tricuspid and mitral valve points, although it is generally difficult to determine the relative loudness of the mitral and tricuspid first sounds. Accentuation of either may be heard and inferred because of greater distinctness at the base as well as the apex, so much so that the first sound at the former region may seem louder than the second sound, provided, of course, that the second sound is not abnormally weak.

Increased intensity of the *mitral* first sound, while usually due to *hypertrophy* of the *left ventricle*, as from chronic interstitial nephritis and arteriosclerosis, aortic stenosis and aortic aneurism, the converse is not necessarily true; that is, in some of these cases the sound may, on the contrary, be quite muffled.

Individual accentuation of the *tricuspid* first sound is less common than of its fellow. When detected, however, it indicates some obstruction to the flow of blood into the left chambers of the heart, directly because of valvular difficulty, or indirectly because of some obstructive condition in the lungs, as *emphysema*. It often happens, nevertheless, that accentuated tricuspid closure lasts but a short time, regurgitation ensuing to relieve the intraventricular pressure, an accentuated pulmonic second sound continuing, however, as in most instances of mitral and aortic lesions, where there is considerable damming back of blood into the pulmonary circulation.

(4) DIMINISHED INTENSITY OF THE FIRST SOUND.—This occurs in all cases of weakness of the ventricular walls from *myocarditis, dilation, or atrophy*, as in the production of weakness of both sounds. But as any weakness of the ventricular muscle will influence the muscular component of the first sound accordingly and primarily, it is most important to estimate the character of the first sound at the apex, as likely to manifest the first evidence of diminishing strength of the left ventricle. This may be watched to develop in the course of a typhoid-fever case, as an example of *absolute* muscular feebleness due to the granular degeneration which takes place in the heart muscle. Or, as the result of a *relative* condition, when hypertrophy fails to sustain a previous compensation, and dilation ensues, the early sign again is a weak, short, flapping character of the first sound (mitral), with a distinct, sharp, valvular quality. Other general and local causes of weakening of the first sound are *chronic wasting diseases, anemias*, etc., and *fatty heart*, whether due to fatty degeneration of the ventricular muscle or to deposit and infiltration of fat upon and between the muscle fibers; chronic myocarditis, especially mitral insufficiency among valvular disorders, and pericarditic deposit upon the heart.

Apparent cardiac causes for weakened systolic sound at the apex may be due to emphysematous lung, pericardial or pleural effusion.

A word of caution is necessary here. Sometimes the first sound is almost inaudible, and the second sound alone being heard with distinctness at the apex may seem to be accentuated when it is really not so. In extreme cases the first sound is completely inaudible, so that nothing but the clickings of the second sound, with alternating silences of more or less decided duration, are heard.

Vierordt refers to the diagnostic value of marked weakening of the apical first sound in cases of *aortic insufficiency*. He explains this as due to "the reflux from the aorta, with the normal afflux from the auricle filling the ventricle abnormally full; it becomes dilated, and thus the tips of the mitral valves, even before the beginning of the systole, are somewhat pushed up. When the systole takes place, there is then only a moderate increase in its tension."

Diminished intensity of the first sound in the *tricuspid* area is noted in all those conditions mentioned before which affect the strength of the ventricular muscle, such as the pyrexial states, degenerative changes, obesity, various forms of chronic auto-intoxication and malassimilation, etc.

(5) ACCENTUATION OF THE DIASTOLIC SOUND. *The Aortic Sound.*—Normally the second sound is better heard at the base than at the apex; consequently, whenever it is well or loudly heard over or near the latter region, the first sound at the same time not being weakened, it means accentuation. Obviously, intensification of the aortic second sound indicates a degree of recoil pressure within the aorta causing the valve leaflets to close with increased tension and suddenness. As, in middle life, the aortic second sound is but a trifle louder than the pulmonic, it is difficult to detect slight accentuation of the former because of the lack of any standard of invariable intensity. In the words of Balfour: "It is only when some distinct quality is superadded, such as that which is very fairly expressed by the word *booming*, that we can speak with perfect confidence . . . that whenever an aortic sound is heard possessing this quality, some degree of dilation of the ascending aorta is always present, or if the aorta be not found actually dilated after death, it is flabby and dilatable—the *sine qua non* for the production of an accentuated aortic sound being the presence in the ascending aorta of a column of blood greater and heavier than usual."

Increased intensity of the aortic sound from increased tension of the aortic circulation may be due also to the following conditions: those which cause increased peripheral resistance to the blood onflow, such as *atheroma of the aorta*, especially arteriosclerosis, *chronic arterial hypertension* (Cook), and *chronic interstitial nephritis*; *aortic aneurism*, and the *cardiac hypertrophy and dilation* which usually accompanies and results from the preceding. Again, we may have accentuated aortic sound without permanent changes in the arteries, and thus the transient increased pressure within the aorta pointing to the beginning of disease, as in association with chill, epilepsy, nervous shock, serous membrane inflammations, acute infections, and at the onset of complications, as of nephritis in scarlatina.

Accentuated aortic second sound has a loud clicking quality, sometimes almost metallic, or it may be low pitched and resonant, as in dilation of the aorta. So sharp is the accentuation frequently in cases of aortic aneurism that it becomes transmissible as the "diastolic shock" to the palpating hand. If marked weakening and dilation of the left ventricle—"heart failure"—occurs, the accentuation subsides and disappears promptly.

As the normal aortic second sound is distinctly louder in those past forty or forty-five years of age, and decidedly so in those of sixty and more years, care should be observed not to misjudge a pathologic condition when there is none simply because accentuation is discovered in individuals at this period of life. The student should be reminded, also, of the possibility of an abnormally loud aortic sound resulting from exposure of the first part of the arch of the aorta by a retracted right lung, or from the consolidation of the portion of lung, or a cavity adjacent to it.

(6) Weakening of the Aortic Sound.—Diminished intensity of the aortic second sound indicates weakness of the heart, temporary, as in the course of infectious and exhausting fevers; permanent, as from the various causes of chronic myocardial degeneration, metabolic, alcoholic, toxic, and the like. In these cases the systolic sound is simultaneously weakened. Intra-aortic blood pressure may be low, as indicated by feeble aortic sound, because of relaxation of the peripheral arteries, or because of a diminished volume of blood thrown into the aorta, as in the stenotic or obstructive lesions of the mitral and aortic valves. In the former of these valvular defects—*mitral stenosis*—the flow of blood to the left ventricle being impeded, each systole propels a diminished volume; in the latter—*aortic stenosis*—the intraventricular volume of blood may be normal, or even slightly increased, but its freedom and fulness of onflow is restricted. Mitral regurgitation similarly lessens aortic blood volume.

Weakened intensity of this sound may result also from loss of blood mass, as in *severe hemorrhages, colliquative diarrheas, anemia*, etc., and from such changes in the valves themselves as cause loss of flexibility, from swelling, thickening, and distortion, the leaflets being unable to recoil with any acoustic intensity.

Finally, in some cases of *aortic insufficiency* the defect is so great as to totally obscure the sound of imperfect closure, nothing but the soft swish of the regurgitating current of blood being heard at the beginning of diastole.

(7) Accentuation of the Pulmonic Sound.—This occurs with any pulmonary or cardiac disease which increases blood pressure within the pulmonary circuit by obstruction to the blood flow in the arteries, veins, or capillaries. Years ago Skoda pointed out that persistent accentuation of the pulmonary second sound was an important aid in the diagnosis of lesions of the mitral valve, although, if congestive or emphysematous disease of the lungs can be excluded, this physical

sign may be indicative of any other cardiac valvular lesion at some stage of its development or secondary influence.

A contributory and concomitant factor in the production of accentuation of the pulmonic second sound is, of course, *hypertrophy of the right ventricle*, since this invariably results from a damming back of blood in the lungs, as in *stenosis or insufficiency of the mitral orifice*, or from *obstructive pulmonary disease*, such as *emphysema, chronic tuberculosis*, and *chronic fibroid pneumonia* (*phthisis*). So long as the accentuation is maintained to a degree of intensity equal to or greater than that of the aortic sound, it is a sign of competency of the right ventricular muscle, and the absence of insufficiency with regurgitation at the tricuspid orifice.

The last point mentioned is of extreme diagnostic importance in cases of acute lobar pneumonia, where the danger of failure of the right heart is so constant and arises so suddenly. In emphysema, notwithstanding the dilated lung overlaps the heart to a greater extent than normal, the accentuated pulmonary second sound may usually be heard most distinctly of all the valve sounds. In mitral obstruction the pulmonary blood pressure is heightened on account of the overfilled left auricle and pulmonary veins, thus increasing the resistance in the pulmonary arteries and right ventricle, while in mitral insufficiency the back flow of a portion of blood with each systole, meeting that coming from the lungs, in time makes itself felt in the pulmonary artery, with consequent accentuation of the pulmonary valve closure.

In not a few individuals of sedentary habits, an accentuated pulmonic second sound may be apparent only, and due to a comparative weakness of the aortic second sound because of flabby, feeble intra-aortic blood pressure (Hardy).

(8) Weakened Pulmonic Sound.—This is especially important in its relation to previous accentuation, in the course of valvular disease of the heart, as a matter of prognosis. The latter is unfavorable or bad in direct proportion to the degree of diminution of intensity of an accentuated pulmonary second sound. It means that the previously compensating, hypertrophied right ventricle is giving way to a flabby weakening, with dilation, and probably regurgitation at the tricuspid orifice, because of the dilation and relative insufficiency of the valve cusps. As a sign of imminent danger of failure of the right ventricle in that treacherous disease—*pneumonia*—weakening of the accentuated pulmonic sound is of first importance, although, as will be pointed out shortly, the chan

in pitch of this sound may be prompter evidence than change of intensity.

Pitch.—Changes in the pitch of the first or second heart sounds go hand in hand, practically, with those pertaining to quality. Thus, the more the first sound becomes like the second in quality the higher in pitch it becomes, naturally, and the weaker the ventricular muscle is indicated thereby.

It is with the pitch of the second sound, however, that more practical value is connected. As intimated before, this attribute, as an independent and correlated factor, has been neglected as regards the second sound of the heart. At the outset, the qualifying terms of pitch should be used with discrimination, the word accentuation (intensity) not being synonymous with elevation (pitch); in fact, the reverse being true, as a rule, the louder sound being lower in pitch, other things being equal. In the careful auscultation of more than 500 normal, healthy adult individuals, I found that approximately eighty per cent had a slightly higher pitch of the aortic as compared with the pulmonary valve sound. Hence, any rise in pitch equaling or exceeding that of the aortic second sound may be considered as significant of increase of tension within the pulmonary artery, due either to resistance of the blood-flow in the left heart or to some congestive condition in the lung, leading to or already accompanying incipient hypertrophy of the right ventricle. This sign appears before accentuated loudness is decidedly noticed, in most instances. Even later, as the cause of the intravascular increased pressure is maintained and continued, with accentuation of loudness, the pitch also being elevated is no contradiction to the physical law of inverse ratio of intensity to pitch, but simply shows that the law as to pitch supervenes, namely, the greater the tension the higher the pitch.

From which it will be seen that, given a seriously ill case of acute lobar pneumonia, for example, with marked elevation of pulmonic sound pitch, indicating that the right ventricle is meeting the tremendous demands made upon it by the consolidated lung area, the earliest and slightest perceptible depression of that pitch must be no less than the veriest danger signal of beginning failure of that muscle. If the lowering of pitch be but moderate, to a degree corresponding with the aortic, the partial yielding of the ventricular walls which this signifies may fall within the prognostic pale of hope, notwithstanding a justifiable anxiety; whereas, a depression of pitch to the point which is consistent with health means, in this case, a feeble, flabby, fatal, unconditional surrender to the resisting forces and poisoning influences.

Likewise, the incipient development of emphysema—a type of the chronic pulmonary disease—in cases of recurrent attacks of bronchitis (the "winter cough" of old people), and paroxysms of asthma, may be recognized in gradual and persistent elevations of the pitch of the pulmonary valve sound, even before any alterations of the percussion and breath sounds over the lungs may be observed. Here the rise of tension within the pulmonary circulation is explained as due to the resistance to the onflow of blood within the myriads of pulmonary capillaries whose caliber becomes diminished by the overstretching to which they are subjected as the containing alveolar walls become dilated—emphysematous—and, perhaps, also thickened by some fibrosis from the chronic irritation of the causative affections. Dependence upon the element of pitch may be a necessity as well as an independent factor in this matter of diagnosis, since mere accentuation of the pulmonic second sound may be obscured by the buffer, non-conducting effect of enlarging and border-extending lung. A lowering of the pitch of the pulmonic sound in evident emphysema would indicate, therefore, weakening of the right ventricle under the persistent and progressive strain, and the impending congestive disorders which are entailed thereby.

Altered Rhythm of Cardiac Sounds.—Arrhythmia may be due to (1) alterations in the *relative length of the silences* between the two heart sounds, or to (2) a *doubling* or *reduplication* of the sounds.

(1) FETAL RHYTHM OR EMBRYOCARDIA.—Whenever the triple rhythm of the normally acting heart—the "one-two-three," or 3 : 4, or waltz time in music—corresponding to the first and second sounds and the diastolic or long pause, is disturbed so that the latter element is shortened, then the silences are equal in length, and the two sounds follow each other as regularly as the ticking of a clock. The first sound also resembles the second sound so closely that the *tick-tack* has been likened to the equality of the fetal heart-beats; hence the term *embryocardia*. The rhythm is then changed to the 2 : 4 time (Butler). The heart action is at the same time much accelerated and decidedly weakened. Indeed, embryocardial rhythm is directly indicative of a serious weakening of the cardiac muscle, such as occurs in the later stages of the *acute infectious fevers*, in typhoid especially, in *chronic myocardial degeneration*, loss of compensation in *valvular heart disease*, conditions of *collapse*, and in *exophthalmic goiter*.

(2) PROLONGATION OF THE DIASTOLIC SILENCE.—This is usually accompanied with accentuation of both cardiac sounds, the long pause

occupying as much rhythmical time as the third and fourth beats of 4 : 4 musical time. Prolonged diastolic pause indicates either the excessive or cumulative action of digitalis, or the strenuous, "spasmodic effort of an overworked, weak, or degenerated heart to continue its labors."

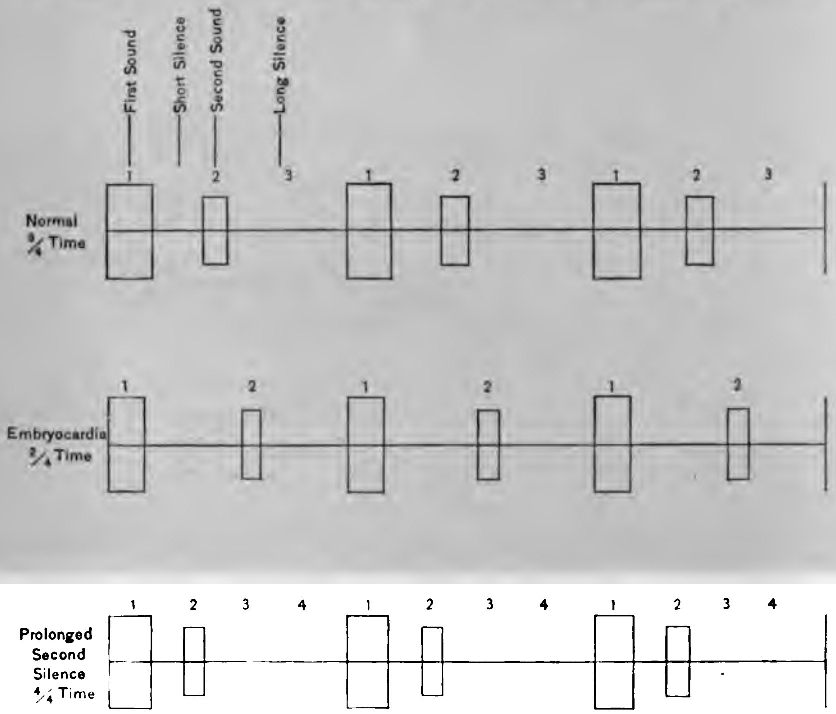


FIG. 56.—DIAGRAM REPRESENTING TWO VARIATIONS FROM THE NORMAL CARDIAC RHYTHM. To be read from left to right. (Butler.)

(3) CANTER OR GALLOP RHYTHM.—The auscultator recognizes a third interpolated or accidental sound as well as the two regular cardiac sounds, the three sounds simulating the hoof strokes of a galloping horse; hence the term applied to this form of arrhythmia. The extra sound occurs in the long silence; in some instances, immediately after the normal second sound, which may be accentuated or not, or in the middle of diastole; in other cases, near the end of diastole, or just before the first sound—at presystole. The sounds are usually similar, short, and partly ringing, or the third sound may be of a different character, like the clack of a cantering horse, which led the Frenchman, Bouillaud, to first apply the descriptive phrase,

bruit de galop. The third sound may resemble either the first or second sound, and may thus seem to be a reduplication of either, respectively.

The gallop rhythm generally indicates a grave, often fatal, condition of *cardiac weakness,* where the sign is persistent. It means that there is a dangerous abnormal increase in the intraventricular blood pressure, and consequently of the tension of the ventricular wall. The more rapid the gallop rhythm the worse the prognosis. According to Babcock, a rapid gallop rhythm differs from a slow, cantering one in that the accent falls most sharply on the first or third instead of the middle one of the three sounds in the former case.

This variety of arrhythmia is an important danger signal in such diseases as *chronic interstitial nephritis, arteriosclerosis,* especially in the *hypertrophic dilation of the ventricles* with loss of compensation; in *myocardial degeneration* from various causes; in *adherent pericardium* with dilation of the heart; and sometimes in cases of *severe anemia.* It is a very grave sign occurring in the course of certain *acute infectious diseases,* as typhoid fever, lobar pneumonia, and acute articular rheumatism, and may be the first sign of beginning *paralysis of the heart in diphtheria.* It is heard more distinctly over the right ventricle in cardiac and pulmonary diseases which cause dilation of this chamber. Thus, the overdistention may result from mitral stenosis, advanced mitral regurgitation, and pulmonary emphysema.

(4) REDUPLICATION OF THE HEART SOUNDS.—When permanent, doubling of the first or second sound is practically always pathologic. Probably in the majority of instances the gallop rhythm is really due to a reduplication of either one of the systolic or diastolic sounds.

Reduplication of the Diastolic Sounds.—Obviously, this is due to broken synchronism of the closing action of the cusps of either the aortic or pulmonic valves. As intimated before, reduplication may be transient or physiologic, and when not so its diagnostic value may be quite subordinate to other associated physical signs. It is produced directly by an abnormal increase of pressure within the peripheral or pulmonary circulation, thus throwing added strain upon the left or right ventricle, respectively. The interval between the doubled sounds is usually extremely short, so that they may be said to run together, whereas in the canter rhythm, a distinct, complete interval is demonstrable. In doubtful cases of doubling of the diastolic sounds, the phenomenon may be elicited better by noting it at the end of inspiration and the beginning of expiration.

Owing to the normally greater pressure within the aorta as compared to that within the pulmonary artery, the aortic valve closes a fraction of a second earlier than the pulmonic, but this is not appreciable to the ear. In reduplication, the second sound may be illustrated by pronouncing the syllables lub-tupptup instead of the normal lub-tup, the first of the two diastolic sounds being accentuated somewhat, and often slightly elevated in pitch. Balfour likens the reduplicated sound to that made by "a hammer which strikes the anvil, rebounds, and strikes again, remaining motionless."

The frequent association of reduplicated second sound with *mitral stenosis* makes this sign have some diagnostic corroborative value. The tension in the pulmonary artery is raised to such a degree because of the mass of blood held back by the mitral obstruction, so that, as explained by Ceradini, the blood pressure is sufficient to separate the sound of closure at the end of cardiac systole from that produced by the arterial systole—in health, these two events being heard as one diastolic sound. Next to mitral stenosis, the most frequent causes of reduplication of the diastolic sound, from exaggerated pressure in the pulmonary circulation, are the following: emphysema, tuberculosis, and bronchopneumonitis; hypertrophic dilation of the right ventricle.

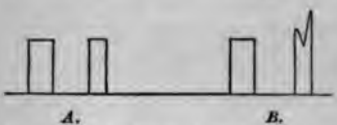


FIG. 57.—*A*. Normal first and second sounds. *B*. Reduplicated and accentuated second sound. (Gibson and Russell.)

Reduplication of the second sound is most clearly heard near the middle of the sternum, or at the left border of the sternum between the third and fifth costal cartilages; in cases of mitral stenosis it is often heard at the apex. If the cardiac action becomes rapid, the reduplication may not be distinguishable, or may persist as a short murmur.

Apparent or *false doubling* of the second sound has been pointed out by Sansom and other English clinicians. This phenomenon is also peculiar to cases of *mitral stenosis*, and may be heard at the apex, while at the base accentuation of the second sound without reduplication may be heard. This simulated doubling frequently passes into a distinct diastolic murmur. Its explanation is most plausibly given by Sansom. He holds that the normal physiologic second sound is followed by an interpolated third sound due to the sudden tension of the mitral curtain from the impact of the blood coming from the auricle to the ventricle, at the beginning of diastole. It will be recalled that the blood is under considerable pressure in the left auricle in cases of mitral obstruction, and its sudden release into the dilating ventricle comes with a decided shock against the mitral cusps immediately after the pulmonic valve closure.

Reduplication of the diastolic sound due to increased tension within the aorta is less commonly met with, as in advanced cases of chronic arterial hypertension, with or without arteriosclerosis, and in chronic Bright's disease.

Reduplication of the Systolic Sounds.—Pathologic doubling of the first sound is comparatively rare, more apparent than real, and may be an incipient presystolic murmur preceding the actual first sound by an extremely short interval. The doubling is not due to a synchronous contraction of the ventricles, but most probably to non-synchronous closure of the mitral and tricuspid valves, or to non-synchronous tension of the cusps of these valves.

Under any explanation, the physical sign indicates excessive pressure within the ventricle—usually dangerously dilated—over which the doubling is best heard: over the right ventricle in mitral lesions and obstructive pulmonary diseases, over the left ventricle in chronic nephritis, especially, with increased resistance in the peripheral arteries and cardiac hypertrophy.

(5) IRREGULARITY.—When the cardiac sounds succeed each other with a variety of divergences as to rapidity, quality, loudness, and relative lengths of the intervening silences in several or all of these characters, the simple term *irregularity* is applied to them. Thus, uniformity of rhythm may be broken by a number of combinations of these changed attributes, the most common, however, being those of strength, duration, and frequency, one or two distinct sounds rapidly following each other with short intervals, to be followed in turn by a longer silence and a few weak, abortive sounds; or these features may be inverted, etc. The gallop rhythm is really a form of irregularity. Gravity of myocardial condition is indicated by irregularity of the cardiac sounds; this is especially marked in cases of mitral disease with failure of compensation, in fatty heart, and in severe debility during or following acute toxemic febrile diseases.

(6) INTERMITTENCY.—This may occur either as a subdued and abortive, or as a totally suppressed sound at certain intervals—a sort of regular irregularity. The intermissions may be one in five, seven, ten, twelve, or eighteen beats, and so on, as the case may be. The cause is often obscure, and as often is unconnected with any evident pathologic condition of the heart. It may be a temperamental peculiarity, a neurosis, or a manifestation of a dyspeptic, gouty, autotoxic, or nephritic affection.

CHAPTER XIII

AUSCULTATION (Concluded)

ADVENTITIOUS SOUNDS OR CARDIAC MURMURS

THE abnormal or adventitious sounds produced under the precordial area may originate within the heart, and are then termed *endocardial*; or they may come from the pericardial sac, and are thus known as *exocardial* or *pericardial*. *Pleuropericardial* and *cardiopulmonary* sounds may also be heard in the region of the heart overlapped by lung or adjacent thereto. Although the word *murmurs* is used by some systematic writers to embrace all adventitious sounds heard in connection with the heart's action, practically it is limited to include only those which are endocardial; that is, produced within the cardiac chambers and beginnings of the great vessels.

(A) CLASSIFICATION OF MURMURS

The *endocardial* murmurs are subdivided into (1) *organic* or *structural*, and (2) *functional* or *dynamic*, *hemic* or *anemic*. The latter class are also designated frequently as inorganic and accidental; they are associated with disorders, more or less temporary, affecting the quality or composition of the blood, such as the essential and symptomatic anemias, debilitating chronic diseases, etc., or with disturbances of cardiac innervation.

The organic endocardial murmurs are valvular in origin; that is, they are due to some structural defect, distortion, obstruction, or other pathologic alteration of the cardiac valves or orifices. While, therefore, murmurs are nearly always indicative of certain valvular changes, yet, in the words of Balfour, "their evidence must be accepted with caution, first, because we may have murmurs of exocardiac origin which simulate very closely those of valvular origin, and, second, because murmurs truly of valvular origin may disappear temporarily or permanently. Thus, we may have a murmur apparently of valvular origin which is really exocardiac; second, we may have a murmur truly of valvular origin, yet without valvular lesion, which may disappear, leaving the heart uninjured; and, lastly, we may have a murmur truly of valvular origin which may disappear temporarily or permanently, the valvular lesion still continuing." Hence, a murmur must never be considered a pathognomonic factor in physical diagnosis, but always correlated and estimated in connection with the results obtained by inspection, palpation, and percussion, as well as the auscultation of the normal heart sounds.

The abnormal sounds of pericardial origin are usually termed *friction sounds*; likewise those of pleuropericardial production. The cardiopulmonary sounds are designated as murmurs, probably because of their softer quality, similar to the endocardial murmurs commonly encountered.

Aneurismal murmurs, or *bruits,* are usually heard over the aortic arch and at the base of the heart.

(B) MODE OF PRODUCTION OF MURMURS

The *physical explanation* of murmurs may be rendered more lucid by the appended preliminary remarks relating to the factors involved. In the first place, it should be mentioned that formerly it was held that endocardial murmurs resulted directly from the friction caused by the passage of blood over roughened surfaces and constrictions of the endocardium. The only tenable view, that now held, attributes the production of murmurs to eddies or currents of blood set up by obstructions at the orifices or leakages at the valves, these disturbances in the blood flow producing the sound vibrations which are conducted to the surface of the chest. Normally, the blood flows through the valvular orifices, in the physiologic direction and rhythm, without sound.

(1) However, it is with the blood as with any other liquid under a certain degree of pressure, namely, that whenever it passes or is forced through a constriction of its containing tube into a wider portion beyond, audible vibrations are generated by the eddies and currents thus set up. These secondary currents are the "*fluid veins*" of the physicists. In the diseased human heart there may be a narrowed orifice leading into a larger normal or dilated cavity; or the orifice may remain normal in caliber, but relatively narrowed because of marked dilation of the cavity. In either case, the fluid veins set up, and their accompanying vibrations follow the main current, producing so-called *direct* or *onward murmurs.* These are the murmurs of *stenosis* or *obstruction.*

If, on the other hand, the valvular mechanism is so altered that perfect closure is prevented when physiologically it should be accomplished, a part of the blood regurgitates through the small aperture into the chamber from which it came previously, as an opposing or backward fluid vein. The vibrations produced are doubtless due partly, also, to the oblique impact of the blood stream against the beveled projecting edges of the incompetent leaflets (Davidson). This valvular *insufficiency*, therefore, gives rise to *indirect, backward,* or *regurgitating murmurs.* According to Geigel, who used mathematical formulæ bearing upon the pitch of murmurs to prove his point, these are rationally explicable only on the basis of transverse vibrations of the cardiac or of the vascular walls surrounding the disturbed blood stream.

(2) The strength or loudness or audibility of endocardial murmurs will depend upon the force and rapidity of the blood flow. Ventricular weakness may cause the previously distinct murmurs of a hypertrophied heart to diminish in intensity, or even disappear entirely.

(3) That a lowered specific gravity and consequent thinner state of the blood permits it to be thrown into vibration by valvular defects more readily is quite probable. In fact, all three of these physical factors may be present in varying degrees in the production and modification of organic murmurs.

Pathologic Conditions.—The principal local lesions which are responsible for the production of murmurs at the valve orifices are the results of endocarditis, which in turn, in perhaps seventy per cent of the cases, is caused by acute articular (inflammatory) *rheumatism*; the other three etiologic disorders of the "big four isms"—as I have often designated them to students—are *alcoholism*, *syphilism*, and *athleticism*. Among the special changes are the following: Adhesion of the flaps of a valve and shortening of the chordæ tendineæ; cicatricial narrowing and constriction of the arterial or auriculoventricular orifices; projection into the lumen of the aorta or pulmonary artery, etc., of vegetations or calcareous plates and the like upon the walls or valves; disease (degenerative) of the papillary muscles; loose portions of ruptured valve cusps, bands, and strings vibrating in the blood stream; vascular dilations, especially aneurismal.

To summarize, the main physical alterations productive of organic endocardial murmurs are as follows:

(1) *Insufficiency*, or incompetency, causing regurgitation of blood when the valves fail to close at the physiologic time.

(2) ***Stenosis*** (constriction), or obstruction at the orifices, interfering with the free and full flow of blood when the valves should normally be open.

(3) ***Relative insufficiency*** at a valve (especially the mitral or tricuspid) orifice, because of dilation of the heart chamber (weakening of the myocardium) containing it, the simultaneous dilation of the orifice causing improper closure of the curtains, which are unable to meet.

Murmurs sometimes occur at *non-valvular orifices*, such as an open foramen ovale or perforated ventricular septum—congenital, rare conditions. Likewise, a patulous ductus arteriosus may be the cause of a murmur.

Characteristics.—It will be seen that murmurs may be of several varieties; they may vary in causation, in combination, and in general and specific characteristics. There may be but one murmur present with distinctive or obscure features, or two or three at different orifices, perhaps two at one orifice (double lesion). Hence, to state the important and significant facts about a murmur which it is the purpose to learn should come first, and then a seriatim consideration of these in detail.

The points to be noted about a murmur are the following:

(1) Its *localization* or area of greatest intensity.

(2) Its *time* or place in the cardiac cycle.

(3) Its *area of conduction* and *direction of transmission.*

(4) Its *acoustic attributes*—quality, intensity, pitch, and duration.

(5) Its relation to the normal sounds of the heart.

(1) LOCALIZATION, OR THE POINTS OF MAXIMUM INTENSITY.—The first step in the diagnosis of a murmur is to localize it, and thus determine the valve or orifice affected. This is ascertained by finding its position of maximum intensity. The points of greatest loudness correspond, with few exceptions, to the areas already described, where the respective valve sounds are best heard; that is, *mitral valve murmurs* are most distinctly heard at or near the apex; *aortic murmurs*, at the right second intercostal space, close to the sternum; *tricuspid murmurs*, over the lower part of the sternum; and *pulmonary valve murmurs*, at the left second intercostal space, close to the sternum. A murmur whose maximum intensity does not coincide with any one of these areas is probably not of valvular origin.

At the *aortic area*, aneurismal bruit may also be heard. To the area, other murmurs than those produced at this e rare, may be transmitted, and the accidental or h are frequently audible here also. The *tricuspid area*, wh n any other, corresponds with the anatomic seat of the orif is not infrequently a region where two other valve murmurs

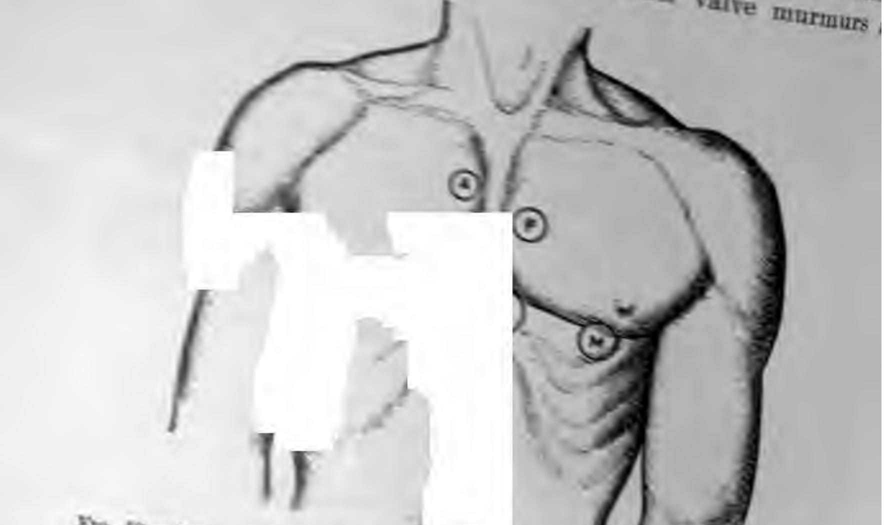


FIG. 58.—SHOWING PRECORDIAL AREAS. *A*, aortic area; *P*, pulmonary area; *T*, tricuspid area; *M*, mitral area. (Gibson and Russell.)

distinctly heard, namely, that of aortic insufficiency and that of mitral insufficiency. The former, diastolic in rhythm, is often more plainly heard here than directly over the aortic area, probably because the downward regurgitating current of blood does not gather audible vibrations so high above the valve in these cases; indeed, this murmur is sometimes as distinct in the left fourth interspace, near the sternum, as in the aortic area. A mitral regurgitant murmur (systolic) of marked loudness may be audible in the tricuspid area. The tricuspid regurgitant murmur (systolic) is the only one of the right-sided lesions which is ordinarily met with, and even it is quite uncommon. In the *mitral area*, the aortic regurgitant murmur may be heard in some instances, because transmitted thither. On the other hand, analogously, the murmur of mitral insufficiency, although less frequently, is well heard, not at the apex but near the left base of the heart—that is, in the third interspace, parasternal line, over the dilated left auricle, into which the regurgitating blood flows.

Auscultation of the heart from the rear **is expedient in cases of valvular affections at times, as the greater the enlargement of the right ventricle the more the apex and left ventricle are pushed toward the back. According to Libensky, dorsal auscultation is particularly useful in differentiating mitral from aortic insufficiency. In the former, with vague anterior signs,** it commonly happens that the systolic **murmur** is best heard at the inferior angle of the left scapula. **When the aortic** valve is involved, however, the dorsal findings **are loudest at the left** supraspinous fossa, and toward the right **and downward to the** third dorsal spinous process.

(2) TIME OR RHYTHM.—By far the majority of organic **valvular murmurs** may be diagnosticated, that is, the lesions producing them **may** be pretty positively inferred upon the double basis of the facts **of the area** of greatest intensity and of the time of the murmurs. **Given** these two physical signs in conjunction with the anatomic and **physiologic** data related thereto, and the pathologic significance is **virtually** a matter of logical deduction with syllogistic regularity. **The** determination of the area of maximum loudness fixes the valve **orifice affected**—the anatomic element; that of the time during the **heart's** cycle when the murmur is heard indicates what the **normal condition** of function should be at that orifice at that time, **and simultaneously** whether the abnormality is obstructive or **regurgitant**, **since** it is either one or the other; and so we have the physiologic **and** pathologic elements.

For diagnostic purposes, the correlation of these elements may be **simplified** and condensed by the statement of two guiding principles, **as follows:**

(*a*) **Murmurs** of *insufficiency* or *regurgitation* are heard at that **moment** or time during the heart's cycle when the *affected valves* ***ought** normally to be closed*; that is, they are heard at *systole* when **the** auriculoventricular or venous (mitral and tricuspid) valves leak, **and** during *diastole* when the arterial (pulmonic and aortic) valves **are affected.**

(*b*) **Murmurs** of *obstruction* or *stenosis* **occur at that time or stage of cardiac** action when *normally blood passes **through the orifices affected***; **that is, they** are heard during *systole* **with disease at the arterial openings**; during *diastole,* when the **auriculoventricular are affected.**

The clinical facts which thus justify and **harmonize with these physiologic** and pathologic principles are actually and obviously the **following:**

pulmonic area, other murmurs than those produced at this valve, which are rare, may be transmitted, and the accidental or hemic murmurs are frequently audible here also. The *tricuspid area*, which, more than any other, corresponds with the anatomic seat of the orifice, is not infrequently a region where two other valve murmurs are distinctly heard, namely, that of aortic insufficiency and that of mitral insufficiency. The former, diastolic in rhythm, is often more plainly heard here than directly over the aortic area, probably because the downward regurgitating current of blood does not gather audible vibrations so high above the valve in these cases; indeed, this murmur is sometimes as distinct in the left fourth interspace, near the sternum, as in the aortic area. A mitral regurgitant murmur (systolic) of marked loudness may be audible in the tricuspid area. The tricuspid regurgitant murmur (systolic) is the only one of the right-sided lesions which is ordinarily met with, and even it is quite uncommon. In the *mitral area*, the aortic regurgitant murmur may be heard in some instances, because transmitted thither. On the other hand, analogously, the murmur of mitral insufficiency, although less frequently, is well heard, not at the apex but near the left base of the heart—that is, in the third interspace, parasternal line, over the dilated left auricle, into which the regurgitating blood flows.

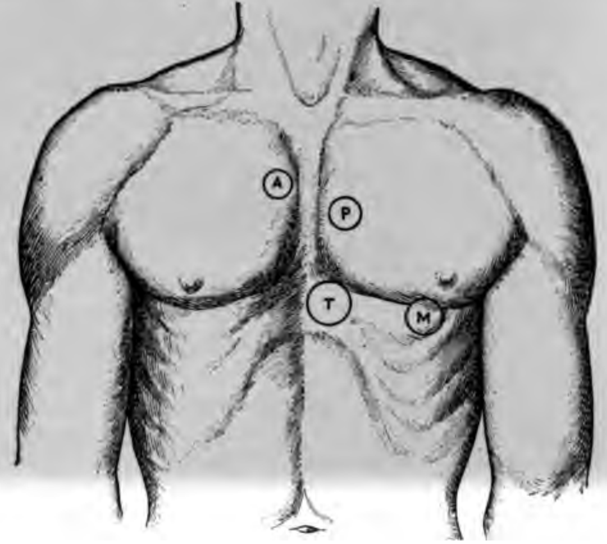


FIG. 58.—SHOWING PRECORDIAL AREAS. *A*, aortic area; *P*, pulmonary area; *T*, tricuspid area; *M*, mitral area. (Gibson and Russell.)

Auscultation of the heart from the rear **is expedient in cases of valvular affections at times, as the greater the enlargement of the right ventricle the more the apex and left ventricle are pushed toward the back. According to Libensky, dorsal auscultation is particularly useful in differentiating mitral from aortic insufficiency. In the former, with vague anterior signs, it commonly happens that the systolic murmur is best heard at the inferior angle of the left scapula. When the aortic valve is involved, however, the dorsal findings are loudest at the left supraspinous fossa, and toward the right and downward to the third dorsal spinous process.**

(2) TIME OR RHYTHM.—By far the majority of organic valvular murmurs may be diagnosticated, that is, the lesions producing them may be pretty positively inferred upon the double basis of the facts of the area of greatest intensity and of the time of the murmurs. Given these two physical signs in conjunction with the anatomic and physiologic data related thereto, and the pathologic significance is virtually a matter of logical deduction with syllogistic regularity. The determination of the area of maximum loudness fixes the valve orifice affected—the anatomic element; that of the time during the heart's cycle when the murmur is heard indicates what the normal condition of function should be at that orifice at that time, and simultaneously whether the abnormality is obstructive or regurgitant, since it is either one or the other; and so we have the physiologic and pathologic elements.

For diagnostic purposes, the correlation of these elements may be simplified and condensed by the statement of two guiding principles, as follows:

(*a*) Murmurs of *insufficiency* or *regurgitation* are heard at that moment or time during the heart's cycle when the *affected valves ought normally to be closed*; that is, they are heard at *systole* when the auriculoventricular or venous (mitral and tricuspid) valves leak, and during *diastole* when the arterial (pulmonic and aortic) valves are affected.

(*b*) Murmurs of *obstruction* or *stenosis* occur at that time or stage of cardiac action when *normally blood passes through the orifices affected*; that is, they are heard during *systole* with disease at the arterial openings; during *diastole*, when the auriculoventricular are affected.

The clinical facts which thus justify and harmonize with these physiologic and pathologic principles are actually and obviously the following:

The mitral regurgitant murmur is systolic; the aortic regurgitant, diastolic; the aortic obstructive murmur, systolic; the mitral stenotic, diastolic, or, as it is commonly designated, *presystolic*, because it is best heard near the end of diastole or just before systole.

With similar lesions on the right side of the heart, tricuspid and pulmonary valve murmurs have the same times, since the physiologic functions on the two sides are homologous.

The mode of reasoning in the physical diagnosis of a murmur may be put forth by two examples; thus, a murmur is best heard at or near the apex (the mitral area), systolic in rhythm; normally, the mitral valve should be closed at this time; therefore, it must leak (insufficiency) in order to cause a murmur at this time. Again, a loud murmur is most audible in the right second interspace, also systolic; the aortic valve is normally open, and blood passing freely through the orifice at that time; hence, there must be some obstruction or narrowing at the orifice to set up a murmur here at this time.

Murmurs are best heard and timed by requesting the patient to stop breathing for a little, so as to exclude the occasional intervention of the respiratory murmur, which sometimes resembles a soft endocardial murmur, and by placing a finger upon the carotid impulse, which is synchronous with the first or systolic sound of the ventricles. (The radial pulse cannot be employed, as it occurs shortly after systole.) In many cases of very weak and rapid heart action, the latter procedure may be absolutely necessary to time a murmur, as the first sound may be indistinguishable from the second by the acoustic qualities alone.

At this point may be considered appropriately (5) the *relation to the normal sounds of the heart* which these various murmurs commonly sustain. The mitral systolic may quite replace the first sound, or it may alter and run off from the first sound, or distinctly follow this sound, in the short silence between it and the second sound (*late systolic murmur*). As a systolic murmur may occur at any one of the four orifices, its localization should be precisely determined. To repeat, the *mitral systolic murmur* indicates that there is leakage at this orifice, the cusps being too small to close the orifice, on account of shrinking, or too stiff to admit of true coaptation, or the myocardium may be degenerated and dilated, so that the cusps are too small to close the dilated opening, or the ventricle too weak to contract the orifice to the natural size of the valve.

The *aortic systolic murmur* usually occupies a distinct portion of the first sound, and of the short interval between this and the second sound. It does not always mean that there is actual narrowing or stenosis of the orifice, or even a limited obstruction by the valve; it may result from a roughening of one or more of the aortic cusps, or from moderate dilation of the aorta just above the valvular region. True obstruction may be due to thickened cusps, vegetative or calcareous growths on their surfaces, or to partial adhesion of the cusps to one another.

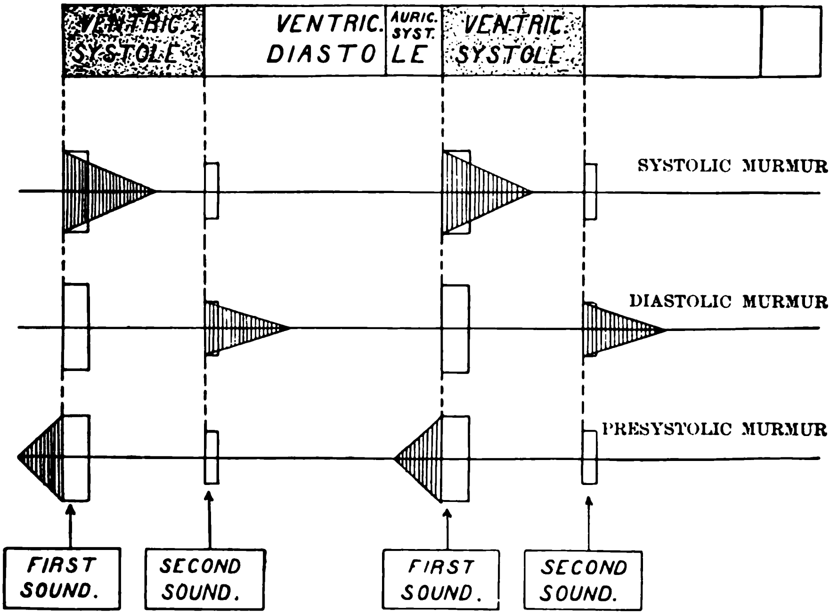


FIG. 59.—DIAGRAM SHOWING THE THREE CHRONOLOGICAL TYPES OF MURMURS AND THEIR RELATION TO THE SOUNDS OF THE HEART. The events of the cardiac cycle are given above for comparison. Murmurs shaded with vertical lines. To be read from left to right. (Butler.)

The *aortic regurgitant murmur* usually begins with and may obliterate the second sound; also occupies the greater part of the diastolic silence. The lesion which frequently produces this murmur is shrinking and thickening of the valves. Vegetations and calcareous nodules may also cause leakage by preventing close contact of the edges of the cusps, and thus forming an aperture through which the blood escapes back into the left ventricle during diastole. Rupture or perforations of the valves, and relative insufficiency from dilation of the aortic ring, may also give rise to an upper or middle sternal murmur of diastolic in time.

The *mitral presystolic murmur*, as its name implies, occupies the end portion of diastole, and ceases with the beginning of the first or ventricular systolic sound. It is rarely heard during the first third or half of the diastolic period. The augmentation of intensity which characterizes it as it approaches the first sound is due to the auricular systolic impulse given to the obstructed flow of blood at the mitral orifice. This murmur is generally due to obstruction or stenosis at the mitral orifice, especially from "adhesion of the anterior and posterior mitral cusps to each other along their lateral edges." A murmur which occurs during the whole of diastole—that is, while the blood should be noiselessly passing from the auricle to the ventricle (*mitral diastolic murmur*)—and before the auricle contracts, may rarely be heard in certain cases where the pressure in the auricle is high and the ventricular suction power vigorous.

The *tricuspid regurgitant murmur*, of all the right-sided lesions of the heart, is the most common. It is usually secondary to advanced mitral disease in which the resultant turgid pulmonary circulation has taxed compensatory hypertrophy of the right ventricle so long or so severely that weakening and dilation ensue, with relative insufficiency at the tricuspid orifice from non-approximation of the cusps during systole. At the same time there may be coincident endocarditis, affecting and distorting the valve, however. This murmur may bear the same relation to the first sound and the succeeding short silence which the mitral systolic does. *Diastolic* and *presystolic tricuspid murmurs*, and the *systolic* and *diastolic pulmonary valve murmurs* occur with extreme infrequency, and are usually of congenital origin, although sometimes due to ulcerative endocarditis. Other lesions of a congenital nature may be associated with pulmonary murmurs, such as patent ductus arteriosus, open foramen ovale, or imperfect closure of the ventricular septum. The systolic murmurs heard in the pulmonic region are more often functional or anemic in causation. The diastolic murmur of pulmonary insufficiency may be relative from persistent high pressure in the pulmonary artery leading to dilation of the orifice, a mitral lesion being the primary cause.

(3) DIRECTION OF TRANSMISSION.—Of the four principal organic valvular murmurs met with, but one, the mitral presystolic, is not transmitted along the lines of the blood current beyond the area of its maximum loudness, although it may have an immediately surrounding area of several inches of conduction. The rule is that murmurs of obstruction are transmitted in the direction of the passing current of blood, and that those of insufficiency proceed along the

lines of the regurgitating currents. The extent of diffusion of a murmur depends upon the quality and strength of the vibrations producing it, the condition of the cardiac tissues near the diseased orifice, the proximity or remoteness of relation of the cavity of the heart in which the murmur originates to the chest wall, and structural variations affecting conductivity in the adjacent tissues, as the ribs, sternum, and the presence or absence of pulmonary consolidations or cavities. The lines of selective propagation of the individual murmurs will be considered separately.

Mitral Systolic Murmur.—This murmur, significant of insufficiency at the left auriculoventricular valve, is transmitted outward—toward the left—and slightly upward. Even a soft murmur may be audible as far as the anterior axillary line, in the fifth interspace. Louder murmurs, besides being heard over the whole precordium, sometimes even to the right of the sternum, are usually transmitted as far as the angle of the left scapula, or two interspaces above the angle, and in exceptional cases even to the right scapula. Obviously, the diffusion of the mitral systolic murmur extends over a wider area when, as often occurs, the apex is displaced downward and to the left, and is in contact with the chest wall to a greater extent, because of hypertrophy of the left ventricle. It should be noted that this murmur may also be transmitted with distinct intensity to the left base of

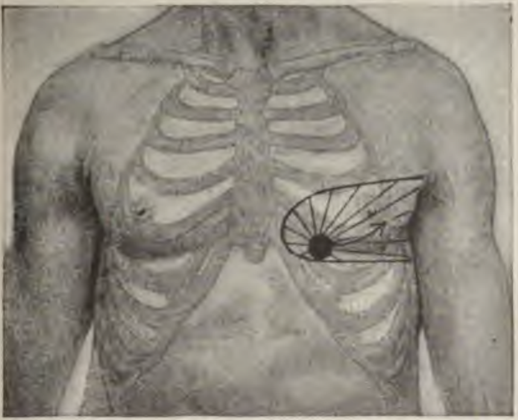


FIG. 60.—MITRAL SYSTOLIC MURMUR. This may indicate mitral insufficiency, anemia, acute infectious disease (myocarditis), left ventricular dilation, malformation of the heart, or acute endocarditis. The circle indicates the point of maximum intensity, the arrow the line of selective transmission. The radiating lines represent the area of audibility. If of sufficient intensity, the murmur may be heard over the entire chest. (Butler.)

the heart, near the left auricle, into which the regurgitating blood flows, especially when that cavity is dilated.

As the reflex current of blood, then, passes inward and backward, we must necessarily trace the vibrations of its course of transmission around the left side of the chest posteriorly. Transmission of the murmur to the right is limited by the deadening effect of the right ventricle.

Mitral Presystolic Murmur.—As intimated before, this murmur is seldom transmitted more than a trifle beyond its area of maximum intensity, just within and above the apex-beat. This is because the direction of the vibrating blood current passing through the obstructed mitral orifice is precisely toward the apex, and to the fact that the weakened left ventricle is remote from the chest wall on account of the intervening right ventricle, which is dilated, flaccid, and also non-conducting. Nevertheless, it is not rare to find a presystolic murmur diffused over a larger area than usual. Thus, its area of audition may extend to the right as far as, or even a little beyond, midsternum; to the left as far as the anterior axillary line, and between the third and sixth interspaces.

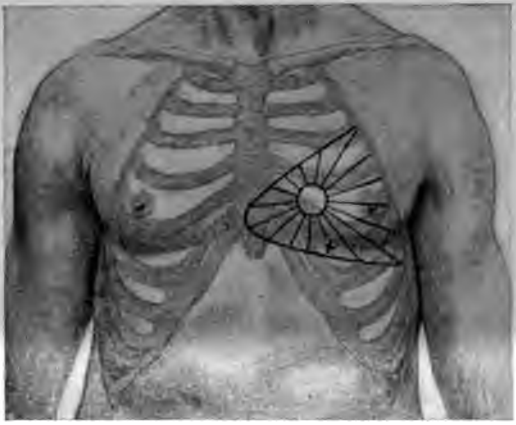


FIG. 61.—MITRAL PRESYSTOLIC MURMUR. This may be caused by mitral obstruction, aortic incompetence ("Flint" murmur), and slight aortic stenosis and adherent pericardium. The circle shows the point of maximum intensity and the usual strict localization of this murmur. The radiating lines represent its *possible* extent of audibility. Apex indicated by the cross. (Butler.)

Although the mitral area presystolic murmur is almost always due to obstruction at this orifice, in some exceptional cases it may really be caused by aortic insufficiency ("Flint murmur"). It is believed

that this murmur is due to the vibrations of the mitral cusps as they are caught between the two currents of blood flowing into the dilated left ventricle, one from the aorta (the regurgitating) and one from the left auricle.

Rarely a presystolic murmur in the mitral area may be produced by aortic obstruction, the first sound beginning slowly and roughly, and by adherent pericardium.

Aortic Systolic Murmur.—This is propagated distinctly over the manubrium sterni, and up into the carotids. It may be heard, also, over the subclavian arteries. As this murmur is often quite loud, it may be heard over a large area of the chest, being conducted, though less audibly, by the sternum, and perhaps by the heart itself, downward and to the left as far as the apex, and may be mistaken there for a mitral systolic murmur unless its area of greatest loudness above and its diffusion into the great vessels of the neck are carefully noted.

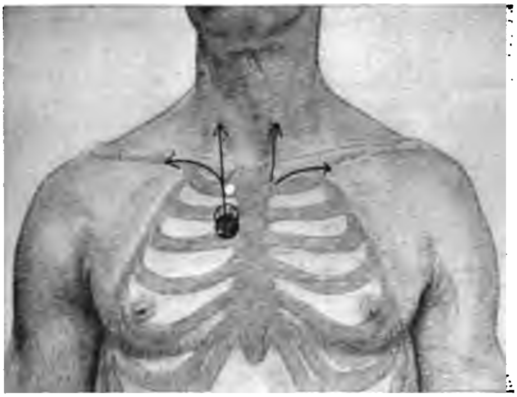


FIG. 62.—AORTIC SYSTOLIC MURMUR. This may be due to aortic stenosis, anemia, dilatation of the aorta, roughening of the aortic segments of the aorta, inequalities of the valve in aortic incompetence, or aneurism of the arch of the aorta. Circles show the points of maximum intensity and the arrows the lines of propagation (into carotids and subclavians) of the murmur. (Butler.)

Aortic Diastolic or Regurgitant Murmur.—This most constant murmur, as regards its lines of transmission, has an area of audibility that extends downward from the middle of the sternum, at the level of the third rib or interspace, and to the left, being heard frequently over the fourth and fifth interspaces from the left sternal border to the apex. This conductivity may be due to the "intimate relation of the aortic cusps with the auriculoventricular septum of the right side of the heart." A diastolic aortic murmur best heard at the apex is held by Foster to indicate an affection of the left posterior cusp of the valve.

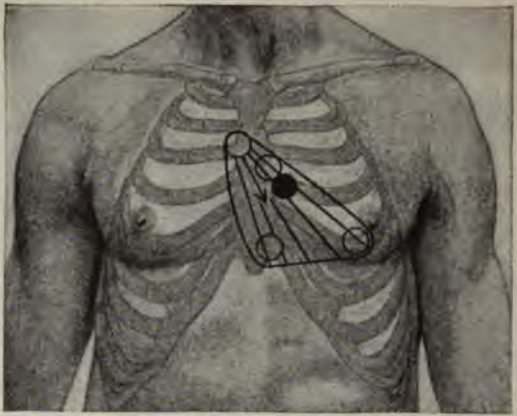


FIG. 63.—AORTIC DIASTOLIC MURMUR. This may be due to aortic incompetency, relative aortic incompetency, or anemia (rare). The solid circle shows the usual point of maximum intensity, the white circles show the occasional points of maximum intensity, and the arrow shows the direction of selective transmission. (Butler.)

Tricuspid Systolic Murmur.—This is the commonest of the right-sided affections, and is usually secondary (relative insufficiency) to extreme tension and pressure within the pulmonary circulation from mitral disease, a previously compensatory hypertrophy of the right

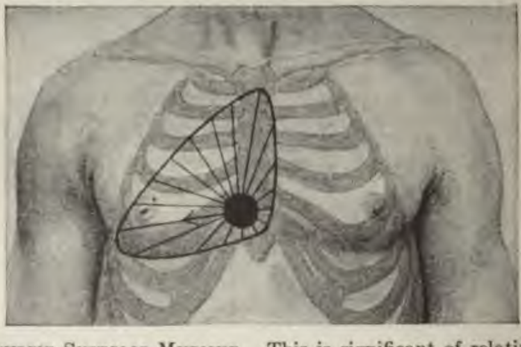


FIG. 64.—TRICUSPID SYSTOLIC MURMUR. This is significant of relative tricuspid incompetency due to dilatation of the right ventricle arising from left-side disease or anemia. The circle shows the point of maximum intensity. It is sometimes heard over the area indicated by the radiating lines. (Butler.)

ventricle giving way to dilation because of the backward stress, and consequently to incompetency of the tricuspid orifice.

The murmur is conducted partly by the regurgitating current and partly by the chordæ tendineæ, upward from the area of maximum intensity at the base of the ensiform cartilage, and to the right as far as the third rib near its sternal junction, and to the left over the area of absolute cardiac dulness toward the apex, the extent of diffusion depending upon the degree of hypertrophic dilation of the ventricles.

Tricuspid Presystolic Murmur.—This infrequent murmur has an area of audition which corresponds practically with the superficial cardiac dulness, and may be propagated to the apex of the right ventricle, although its maximum and total audibility may be circumscribed to the lower end of the sternum.

Pulmonic Systolic Murmur.—This murmur is propagated upward and slightly to the left along the course of the pulmonary artery, from its point of greatest intensity at the left second interspace close to the sternum. As an evidence of congenital malformation of the heart, this murmur is extremely rare; less so as the result of pulmonary stenosis or narrowing from traction or torsion of the artery due to shrinking of the upper lobe of the lung or displacement of the heart. As a sign of anemia, occurring as a hemic or functional murmur, however, it is met with often; similarly, in connection with the excited tachycardiac action in exophthalmic goiter. Even a transmitted mitral systolic murmur or a cardiorespiratory murmur may be heard in this region.

Pulmonic Diastolic Murmur.—This very rare murmur has an area of diffusion downward from the pulmonic (left second) interspace, along the left border of the sternum as far as the fourth interspace. It is not improbable that the murmur is mistaken sometimes for aortic regurgitation. It may occur from congenital malformation of the valve, from ulcerative endocarditis, or from relative insufficiency due to extreme blood pressure in the pulmonary artery.

(4) Quality, Intensity, Etc., of Murmurs.—The *quality* of a murmur differs characteristically from that of the normal heart sounds. While the latter, with their rhythmical "lub-dup," convey the impression of the valve tension of a smoothly acting steam-pump, the former represent the peculiar blowing, swishing, or whistling sounds of the pump with disordered valve action. These abnormal sounds have been likened to the utterance of the various syllables "uf," "uv," "ush," "urr," "b-r-r-r," etc.

As the PITCH and QUALITY of a sound are intimately associated, we may refer to organic endocardial murmurs as low-pitched *blowing*, like the sound of air passing through a large tube ("bellows sound"), or high pitched and whistling or scraping. Many low-pitched murmurs are lightly aspirated, and may simulate closely an inspiratory whiff of adjacent normal lung; others may have a *rumbling, rustling,* or *rasping* quality. The former may be imitated somewhat by whispering the letter "f," or the vowel sounds "oo," "u," "ah," "au," the latter by guttural or growling tones. Some of the higher-pitched murmurs may even be *twanging,* or *musical* and *singing,* in character.

Although none of the qualities just mentioned possess any value in the differential diagnosis of the pathologic nature of the disease affecting the valve or the structures around its orifice, they may have distinct signification of the physical nature of the lesion; that is, soft, blowing murmurs are usually suggestive of regurgitation, while obstructive murmurs are generally rough and grating. Murmurs are not only variable in quality, but a single murmur may change in character because of changes in the size and structure of the orifices and chambers of the heart, in the force of cardiac action, and because of anemic alterations of blood composition.

The qualities of the most common murmurs met with usually are as follows: The mitral regurgitant or systolic murmur has a soft, blowing quality. The aortic obstructive or systolic murmur is harsh, sawing, or filing in character, according to the extent and degree of narrowing and calcareous deposit upon the valve. The aortic regurgitant or diastolic murmur is generally very soft and blowing, often less harsh than the mitral regurgitant murmur, and sometimes hardly audible to the ear of the most careful examiner. Exceptional cases occur, however, where this murmur may be quite rough and vibrant, even musical. The quality of the mitral stenotic or presystolic murmur is distinctive because of the peculiar blubbering, churning sound, rougher than a regurgitant murmur, but softer than the sawing quality of the aortic stenotic murmur. It may be simulated by vocalizing the syllable "rrrb," the last letter terminating abruptly with the immediately following first sound of the heart; so close, indeed, that not infrequently the murmur seems to coincide with this sound, and thus having a systolic time.

The INTENSITY of murmurs is as variable as their quality. It does not depend necessarily upon the severity of the valvular lesions, but may be suggestive often of the character of the lesions, whether causing obstruction or insufficiency. Obstructive murmurs are, as a rule, louder than the regurgitant ones. The loudness of murmurs is more often affected by the force of the heart's action, temporarily, as from excitement or treatment; thus emotion, exercise, and such drugs as digitalis, strophanthus, and strychnine may exaggerate their intensity. Therefore, not only do murmurs vary in intensity in different individual cases, or in multiple lesions in the same individual, but they may fluctuate in connection with a single lesion in auscultating the individual at different times.

(*a*) The *force of the blood stream*, and consequently the strength of the ventricular muscle, is the most important diagnostic and prognostic factor which is indicated by the intensity of a murmur. Naturally, the systolic murmurs are usually louder than the diastolic, other things being equal, because stronger blood eddies are set up by ventricular contraction than by vascular recoil and ventricular dilation. Furthermore, a loud systolic murmur implies hypertrophy and good compensation—a favorable sign; and conversely, if while the case is under observation such a loud murmur becomes gradually weaker, or if the murmur disappears, even, it means that compensation is failing from degeneration and dilation of the cardiac muscle. Under treatment, a previously weakened or inaudible murmur may again assume distinct loudness; so that the mere presence or intensification of a murmur may be a hopeful indication of the condition of a patient's myocardium. Gibson states that the mitral presystolic murmur is particularly prone to disappear and reappear, not only because of the variable energy of the left auricle, but especially because with weakening and distention of the right ventricle the apex is pushed away from the chest wall, and thus prevents the conduction of the sound.

(*b*) The *degree* as well as the *nature of the lesion* affects the loudness of a murmur. As a rule, the narrower the obstructed orifice as compared with the size of the cavity beyond the louder the murmur. Marked aortic stenosis is usually very loud also, because of the added feature of a vigorous hypertrophy to enable the left ventricle to overcome the difficulty in the attempt to maintain a balance of the circulation. Occasionally, however, loud murmurs are heard during life which the slight changes found subsequently post mortem seem hardly related to. On the other hand, hard, calcareous excrescences upon the valve leaflets, as in the aortic disease of very old people, are often accompanied with loud, rough murmurs; these may even be *musical* in some cases, when perforations of valve leaflets may be suspected, or floating shreds of torn leaflets or papillary muscles.

(*c*) *Posture, exercise, and respiration* influence the intensity of murmurs. Of the two principal varieties of lesion, the obstructive murmurs are louder when the patient sits or stands, especially when the auriculoventricular orifices are affected. Regurgitant murmurs, on the contrary, are most distinct when the patient is recumbent. This is often true of the aortic diastolic murmur, although it must be admitted that, as regards posture, the aortic and pulmonary valve murmurs are less affected than are the mitral and tricuspid. Both mitral obstruction and insufficiency may be better elicited by their murmurs while the patient lies flat upon the back. Nevertheless, the rolling, "flowing" sound of a presystolic murmur, which is quite easily heard when the patient is sitting up, may in some cases disappear nearly or entirely when he lies down.

The effects of *exercise* have diagnostic value not only concerning the audibility of the murmur, but also the condition of the heart muscle. A murmur so feebly heard ordinarily that only after a brisk walk of forty or fifty paces, stooping and straightening of the body, and the like, is it rendered most distinctly audible, indicates a lesion of comparative mildness, or a myocardium of developing weakness. On the other hand, marked rapidity of cardiac action following forced exertion may make it difficult to time or even detect a murmur which at a previous examination, with the patient at rest, was patently evident.

The *respiratory period* affects the intensity of a murmur. During inspiration, while the lung is distended as it overlaps the heart, diminished loudness of a murmur is usually noted, especially when the mitral valve is affected. Hence, organic murmurs are better heard at the end of expiration, as contrasted with the functional and cardio-respiratory murmurs, which are more audible at the end of inspiration.

(*d*) The *specific gravity and plasticity of the blood* influences the loudness of a murmur. In cases of anemia with reduction in the number of the corpuscles, and in the fibrin and other proteid percentages (hydremia), murmurs are louder than where these factors are more nearly normal. And thus we find that following a case of severe acute articular rheumatism with blood damage, a mitral insufficiency murmur will be louder than later, when treatment has corrected the anemia.

The DURATION of murmurs differs decidedly in many instances; in some it is short, in others so long as to occupy most of systole or diastole. Practically murmurs always have longer duration than either of the heart sounds. Murmurs of obstruction are commonly of longer duration than those of regurgitation, as it takes longer for the blood current to pass through a narrowed than through a leaking orifice. If the disturbance is sufficiently great, however, and the heart's action sufficiently energetic, an aortic regurgitant murmur may exist throughout diastole, as well as a mitral stenotic. The mitral systolic murmur is usually short in duration, being but a prolongation of the first sound (with roughening), or occupying a portion of the silence between the first and second sounds. Murmurs which replace either of the heart sounds are generally of longer duration than those which occur separately in the short or long silences, and are of correspondingly graver significance.

(C) MULTIPLE OR COMBINED MURMURS

These result from a combination of (usually) two valvular defects, very infrequently three, and with extreme rarity four defects. One should naturally seek to know whether a single or several valvular lesions coexist whenever a murmur is heard over more than one valve area. As the intensity of many murmurs permits of their propagation some distance beyond their areas of maximum audition, and, as shown before, their transmissibility with the direct and regurgitating blood currents being important phenomena, this question as to multiple murmurs must arise almost constantly, whether they be present or not. Indeed, to quote the words of DaCosta more than a generation ago: "How important is it, then, to examine each portion of the heart separately, as much for the purpose of saying what is not as what is deranged."

This problem of diagnosis arises, of course, only when the murmur heard is everywhere the same in rhythm—that is, systolic or diastolic in the various regions; thus, a systolic murmur heard over the precordium may mean a combination of aortic obstruction and mitral insufficiency. Obviously, when both a systolic and diastolic murmur are heard, even at one valve area, or anywhere over the chest, two lesions must be evident either at one orifice, as aortic stenosis and regurgitation, or at two orifices, as aortic and mitral regurgitation.

Where a widely audible murmur is heard, therefore, the difficulty of interpretation may be twofold. *First*, it may be supposed that but one valve is diseased instead of two; that the murmur heard in the second area is merely transmitted from the first. *Secondly,* on the other hand, it may be inferred that there is a combination of two valvular lesions when there is really only one, as when a diastolic murmur heard at the apex is attributed to a mitral stenosis as well as an aortic insufficiency.

To **differentiate** these signs, the appended points in observation may be applied successfully. They are based upon two facts already described, namely, that the various valvular lesions commonly met with have selective areas of maximum intensity, and more or less characteristic differences of quality.

(1) For example, auscultating at the base of the heart, near the third left sternocostal junction, we hear a feeble systolic murmur. Choosing the two valve points, mitral and aortic, for first consideration, we trace the murmur from aorta to apex, step by step, noting changes of intensity and quality in approaching and receding them. Here we note that in approaching the aortic region, over the manubrium and to the second right intercostal space, the murmur becomes louder, harsher, and more widely propagated, especially upward into the carotid arteries. There is no doubt that this is an aortic obstructive murmur. Retracing with the stethoscope, the murmur becomes fainter and softer, though distinctly audible, in the third and fourth left interspaces; in fact, it seems to be a trifle more distinct over the right ventricle, at its left border (fourth interspace outside the parasternal line). In the fifth interspace, over the apex, which is a little outside the midclavicular (nipple) line, the systolic murmur is still louder, yet not so peculiarly harsh as above, and is shorter in duration. It is also heard, tapering off in distinctness, for several inches toward the left axilla. We conclude from the last observation that there is another, a mitral systolic (regurgitant) murmur, because of the two areas of maximum intensity corresponding to valve points, with an intervening space of least intensity for the murmurs best heard at both, and because the character or quality of both these systolic murmurs is different, with lines of propagation in harmony with what we know of either of the murmurs considered singly.

As Gibson points out, another serviceable aid may be found in the fact that "if the murmur is louder at the apex than it is over the left edge of the heart in the space above the apex, or the rib above that, the probability is that there is a mitral as well as the aortic murmur. The reason of this last is apparent, for if the murmur at the apex is the aortic systolic propagated downward by the left ventricle, there is no reason why it ought not to be as audible at any point along the left edge of the heart as at the apex." In cases, however, where the aortic murmur is extremely loud and distinctly conducted over the whole precordium, the presence of a mitral systolic murmur may be inferred by the help of the discovered evidence of elevated tension and pressure in the pulmonary artery, manifested by an accentuation and heightened pitch of the pulmonic second sound.

(2) Again, a soft diastolic murmur is heard at the base, at the topographic-anatomic valve area, also in the right second interspace, with equal but not greater intensity. Approaching the apex, downward and to the left, the murmur becomes rougher and longer in duration, with a maximum of intensity just inside the apex. Is this a case of transmitted aortic regurgitant murmur or one of mitral stenosis, or both? Here the same method of tracing from one valve point to the other, and noting changes in attributes, may be pursued, remembering that the mitral presystolic murmur is not transmitted upward, etc. For further diagnostic data, the signs obtained by the other methods of examination must be correlated, especially in connection with the size of the heart chambers, position of the apex-beat, and the like, as will be pointed out later in describing some of the consequences of the various lesions upon the cardiac walls.

(3) Auscultating near the base of the heart, in a third instance, a systolic murmur is heard with slight intensity. Advancing to the aortic and pulmonic areas with the stethoscope, the murmur is inaudible. Passing downward over the body of the heart, in the parasternal line, the murmur is heard a little better than in the third left interspace near the sternum, but still rather feebly. It becomes distinctly stronger, however, as the apex is approached in the fifth interspace, and is quite well audible for one inch outside the nipple line. Toward the right, in following along the fifth interspace, we notice that the systolic murmur, slightly changed in quality and pitch, also becomes louder until the tricuspid area is reached, where its maximum is heard. That we are dealing with a double valvular abnormality is evident—tricuspid and mitral insufficiency.

The combinations of valvular lesions, as manifested by their accompanying murmurs, are principally the following:

(*a*) Aortic regurgitation and stenosis; mitral regurgitation. We hear the double, to-and-fro or "seesaw" murmur at the aortic area, and a soft systolic murmur at the apex. The aortic second sound is usually obliterated by the systolic-diastolic rushing sounds.

(*b*) Aortic regurgitation and mitral regurgitation. A soft diastolic murmur transmitted from the base to the apex of the heart, followed by a short or systolic murmur transmitted to the left axilla.

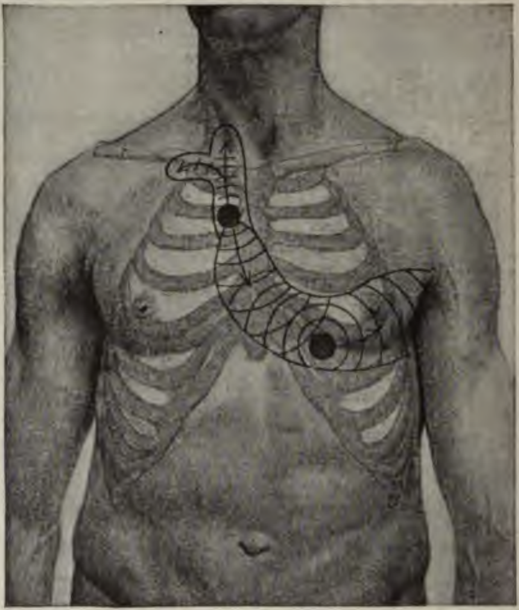


FIG. 65.—COMBINED MURMURS OF AORTIC INCOMPETENCE AND STENOSIS AND MITRAL INCOMPETENCY. (Butler.)

(*c*) Mitral stenosis and regurgitation. A presystolic, churning murmur, ending abruptly with the first sound, and followed by the systolic murmur.

(*d*) Aortic stenosis and mitral stenosis. A rough systolic murmur, heard over the upper portions of the chest, with its maximum intensity at the right second interspace, and propagated into the carotids; then the second sound; then the rolling murmur just before the next first sound, its audition circumscribed around the apex.

(*e*) Mitral regurgitation and tricuspid regurgitation.

(*f*) Mitral stenosis and aortic regurgitation.

(*g*) Aortic regurgitation; mitral stenosis and regurgitation.

(*h*) Aortic stenosis; mitral regurgitation; tricuspid regurgitation.

Tabulated Summary of the Four Principal Organic Endocardial Murmurs

LESION.	AREA OF MAXIMUM INTENSITY.	RHYTHM.	AREA OF AUDITION AND DIRECTION OF TRANSMISSION.
Aortic obstruction or stenosis.	Second right interspace.	Systolic.	Over the upper sternal region; sometimes over the whole precordium; transmitted into carotid artery.
Aortic insufficiency.	Same, or left border of sternum at level of third rib.	Diastolic.	Over lower two-thirds of sternum, and precordium to left of sternum; transmitted to apex.
Mitral stenosis.	Apex of left ventricle.	Presystolic.	Circumscribed to apical region; rarely transmitted.
Mitral insufficiency.	Same.	Systolic.	Over apical and nipple areas; usually transmitted toward axilla; round to angle of scapula; sometimes upward and inward to left auricular region.

(D) FUNCTIONAL OR ACCIDENTAL MURMURS

These are adventitious sounds occurring in certain diseased states, more or less general, in which the cardiac function is disturbed or the circulating blood altered, but in which there is no valvular or orificial anatomic lesion. They are thus found in association with cardiac neuroses, and with anemic and some febrile conditions. The

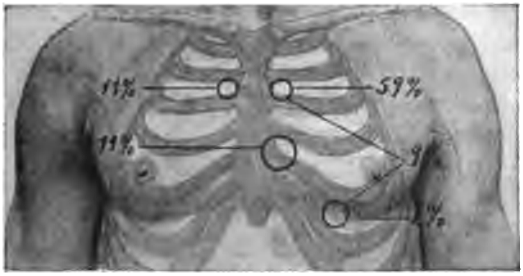


FIG. 66.—SHOWING THE RELATIVE FREQUENCY OF ANEMIC MURMURS AT THE VARIOUS ORIFICES OF THE HEART. Percentages from Sansom. (Butler.)

terms *inorganic* and *functional* are commonly used to designate these murmurs because of the absence of any structural changes affecting the heart. When due to blood states, they are spoken of as *hemic* or *anemic* murmurs. As *dynamic* murmurs they refer more especially to perversions of the regularity, steadiness, poise, or freedom of the muscular action of the heart, probably with some disturbance in the neural function at the same time; these correspond with the cardio-muscular murmurs of Drummond. Perhaps, as Babcock suggests, the term *accidental* is as embracing and appropriate as any, as it " implies that the murmur is a chance result of cardiac action."

Characteristics of Accidental Murmurs.—(1) As to *location*, they are usually best heard at the base of the heart, especially in the pulmonary valve area, although they are not infrequently audible in the mitral area, but seldom in the aortic or tricuspid areas.

(2) In *time*, they are practically always *systolic*.

(3) In *quality*, they are soft and blowing, or bellows-like.

(4) In *intensity*, they are gentle, often feeble; mere soft aspirations, and never harsh, rasping, or sawing.

(5) Their *pitch* is often high, but may be low, and their *duration* short.

(6) Their area of audition is circumscribed, and they are not transmitted beyond the cardiac area.

(7) In some cases, as in chlorotic or anemic subjects, they are accompanied with murmurs in the veins of the neck.

(8) They are usually louder at the end of expiration.

(9) They are not permanent in character, their fugaciousness depending upon the etiology, and varying from a few hours, as the result of hard exercise, to days, weeks, or even months, disappearing whenever general and local improvement ensues.

(10) They are not attended with evidence of cardiac enlargement.

Functional murmurs may be produced by a simple increase in the force and rapidity of the blood current in excitable individuals laboring under temporary physical, mental, or emotional stress, but in whom there is no abnormality of size or structure of the valve orifices or of the valves, nor of the specific gravity or composition of the blood; these are the true *dynamic* murmurs of health, in contradistinction to those due to some irritability or irregularity of the neuromuscular function of the heart, which may, indeed, in certain cases, really be caused by some obscure or latent organic affection of the myocardium, as of patches of fatty or fibroid degeneration.

Production.—The theories to account for the direct origin of the accidental murmurs are so numerous, varied, even antagonistic, and generally unsatisfactory, that one may be justified in taking a more or less personal view based upon the studies and experiences of others, as well as one's own observations and deductions. Hence it is probable, in the first place, that the functional murmurs heard temporarily in those diseases of a pyrexial or wasting nature in which myocardial relaxation or debility is associated, are really the result of a relative insufficiency of the auriculoventricular openings, or of some dilation of the conus arteriosus of one or the other side. In involvement of the tricuspid or mitral orifice, the condition is not always or necessarily a dilation of the opening itself, but may be due to disorder of the function of the papillary muscles and chordæ tendineæ. In these cases there is often a weak, impure first sound at the mitral area, with a short, soft, systolic murmur, best heard at the third or fourth interspace close to the sternum at its left border, loudest at the end of expiration, and in the upright posture.

The fact that many of these accidental murmurs are most distinctly audible in the pulmonic-valve area may be explained by their propagation from their mitral seat of production to the chest wall by conduction along the tip of the left auricular appendix. In the case of temporary dilation of the right ventricle, the sound of relative insufficiency and of an overworking muscle is propagated to the pulmonic area as a tremor (Sansom). It may be also, as Quincke pointed out, that a murmur in the pulmonic area may be produced by a slight squeezing of the pulmonary artery against the chest wall by the dilated ventricle.

In the anemic states—chlorosis, pernicious anemia, and the like—causing the *hemic* murmurs, the condition underlying their production is essentially the same—that is, weakening of the myocardial walls owing to the deficient quality and quantity of blood, imperfect metabolism—a relative insufficiency resulting. The murmurs of anemia are heard usually at the second or third left interspace, near the sternal border, although they may rarely be audible at any of the other valve areas—at the aortic most frequently, then at the mitral and tricuspid.

The precise explanation of the so-called hemic murmurs is difficult, and at this time entirely wanting. Sahli and Vierordt agree in suggesting that they may really be *venous* murmurs transmitted to the heart from the large veins in the thorax which lie behind it. According to Cabot, the diastolic functional murmurs, exceedingly rare as they undoubtedly are, may be "due to sounds produced in the veins of the neck and transmitted to the innominate or vena cava."

Certain non-valvular or functional murmurs may also be caused by congenital anomalies of the heart, and by pressure from deformity outside the heart.

While most of the accidental murmurs are basic in *location*, notably the hemic ones, nevertheless not a few are apical, especially those which may be termed more properly dynamic. Of the first class, chlorosis is a type of disease; of the second, chorea. The chlorotic murmur is often heard in the aortic, and sometimes in the upper mitral areas, but in the former region is not usually propagated into the arteries, except in the most advanced cases. However, as Balfour points out, the functional murmurs of both kinds essentially depend upon simple dilation of the heart from the debility, weakness, and relaxation of the myocardium, the result of the perversions of metabolism and neuromuscular rhythm and power.

Differentiation.—It is often difficult to determine whether we are dealing with a functional or a true organic murmur; and, although the distinctive points of the former have just been given, it may be well to put over against these a comparative summary of those of the organic murmurs.

Functional murmurs are almost always systolic in time; *organic* murmurs are frequently diastolic.

Functional murmurs usually have their areas of maximum intensity at the base, and in the pulmonic area; *organic* murmurs, on the other hand, occur more frequently at the apex, and when found at the base are practically limited to the aortic region.

Functional murmurs are generally very soft, not harsh or musical; nor are they, as is commonly true of *organic* murmurs, transmitted beyond the precordium, or along the lines, especially, of regurgitating blood-currents.

Functional murmurs are usually loudest at the end of inspiration; the *organic*, at the end of expiration.

A *functional* murmur should always be suspected, other things being equal, whenever the sounds are heard in cases of distinct chlorosis or other forms of pronounced anemia, and in neurotic states; an *organic* murmur, whenever there are positive evidences of cardiac enlargement, such as displaced apex-beat, with exaggerated force and extent of impulse, etc.

Finally, the *functional* murmurs differ from the organic in being susceptible to obscuration by pressure with the stethoscope, and to disappearance when the patient assumes the dorsal or right lateral decubitus.

Musical murmurs, as intimated before, may accompany organic valvular, especially aortic, lesions, as whistling, filing, singing, etc., qualities of sound. In some cases they may be caused by sclerotic and calcified aortic cusps, or to fenestrations, or to tears with freely floating ends. Again, the post-mortem findings of musical murmurs have been hard atheromatous plaques in the aorta near the aortic orifice; fibrous bands stretched across the ventricles near the tricuspid or mitral orifices, or, as in a case reported by Potain, a cord between the ventricular wall and the edge of the mitral valve, just below the aortic orifice, and ruptured tendinous cords swinging loosely or forming abnormal attachments.

In the case of the itinerant young Russian, to whom Babcock refers in his book on "Diseases of the Heart and Arterial System," p. 30, and whom I showed to my class in the Medico-Chirurgical College, the scar of a stab wound over the right ventricle suggests that the singing, systolic murmur of his "musical heart" is probably due to tricuspid regurgitation.

Accidental musical murmurs, however, may also occur in certain rare instances. Thus, they have been discovered occasionally in neurotic, slightly anemic young females with tachycardia, and who while under examination have exhibited intense nervousness, and visible and palpable violence of cardiac action. They may be explained as due to the vibration of one of the so-called *moderator bands* or *aberrant cords* sometimes found stretched along or across the intraventricular wall, while under the increased tension of overfilled chamber and extremely rapid action.

Cardiopulmonary or Cardiorespiratory Sounds.—Although strictly within the category of the exocardial sounds (see next topic), so far as their origin is concerned, their presence within and their frequent proximity to the precordium, as well as their mode of production, ally them quite closely to the more purely functional or accidental murmurs; in fact, Potain and others consider all accidental murmurs practically as cardiopulmonary in origin. By this compound term it is meant that the sound is caused by the rhythmic impact of the heart against the overlapping lung, especially that portion known as the lingula.

The cardiorespiratory murmur, which is not at all uncommon, is heard as a short, moderately high-pitched, whiffing sound, quite suggestive of the brusk displacement of air from the vesicles and bronchioles of the surrounding lung. Because intimate contact between the heart and lung is essential to produce this sound, it is natural to reason that it will be most audible at the end of inspiration, fading rapidly with expiration; and so the clinical facts are found to be. Again, the cardiopulmonary sound ceases to be heard when the breath is held. With deep and forcible, though slow breathing, it may be heard throughout the respiratory act, but always louder during inspiration. At other times, during moderate breathing, one hears three or four distinct and rapidly diminishing puffs; then an expiratory silence. It is systolic in time, as a rule. Lying upon the back or the right side usually diminishes the intensity of this sound.

The area of audibility is limited to the apical and left-upper border regions. The sound is not transmissible as are the valvular murmurs, although in rare instances it has been heard under the left clavicle and scapula. It may be modified by external pressure, as with a stethoscope, in the vicinity of its production; either quality or intensity, or both, may be altered. Although the cardiopulmonary sounds may occur under entirely normal conditions, it is not unlikely that in some cases there is a localized or compensatory emphysema against which the heart impinges in its movements. Finally, it should be borne in mind that these sounds are extremely variable, and may be heard during any physical relation of heart to overlying lung sufficient to generate them, without regard to respiration or posture, although those points mentioned above are, in the majority of instances, characteristic.

EXOCARDIAL SOUNDS

The adventitious sounds produced outside and yet associated with the heart, although not usually heard beyond the precordial borders, are mainly those originating within the pericardial sac. Others, of less frequent occurrence, may be pleuropericardial, vascular, pleural, pulmonary, and subphrenic. Excepting the vascular murmurs, they are all characterized by marked variety of quality, indefiniteness of relation to the cardiac cycle, and variability of signs generally.

Pericardial Murmurs or Friction Sounds.—As these names clearly indicate, they represent the effects of the rubbing or friction of inflamed, roughened pericardial surfaces, analogous to pleuritis, since normally these serous membranes, visceral and parietal, glide over each other smoothly and noiselessly.

Attention will be directed at the outset to the peculiar QUALITY of the pericardial friction sounds. They are variously described as grazing, grating, scratching, often brushing, shuffling, scraping, creaking like leather, rustling or crumpling like silk or parchment, crunching like snow—all depending more or less upon the dryness of the surfaces, or the viscosity, thickness, and chronicity of the exudate.

As to LOCATION, pericardial friction is best heard over the body or center of the heart, near the roots of the great vessels, and over the base of the right ventricle, seldom at the apex. In other words, it is found usually in the third and fourth left interspaces, where it does not coincide with the areas of maximum intensity of the organic valvular murmurs. Of course, it may be heard over a large part of the precordium in cases of extensive fibrinous pericarditis; often in the second interspace, and nearly as far as the left nipple.

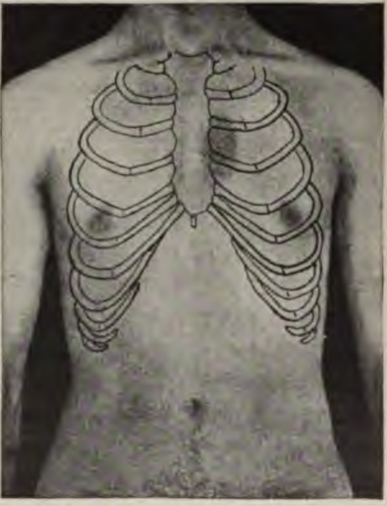


FIG. 67.—USUAL LOCATION OF PERICARDIAL FRICTION SOUND AND FREMITUS. (Babcock.)

It is *circumscribed* rather sharply—that is, its audibility is limited pretty closely to the point of its origin—and it is not propagated along any blood-current.

RHYTHM.—This is the most important physical sign to be determined in connection with the pericardial rubbing sound. In the first place, it is notoriously variable and changeable in time. However, it is mostly to-and-fro, systolic-diastolic, but may occur in any portion of these cardiac functions; it may accompany and overlap, or go between, preceding or following either or both heart sounds, more often being interposed between than synchronous with them. Although most frequently associated with ventricular systole or diastole, sometimes, as I have heard in two cases, one fatal, there may be a triple friction sound due to auricular systole, like the 3:4 rhythm of waltz music. A double sound may, so to speak, hug the second sound.

A characteristic feature of the pericardial murmur is its irregularity of rhythmic relation to the systole and diastole, being at one time closer to one, then in a day or two to the other corresponding sound.

INTENSITY.—The factors which influence the loudness of the pericardial rub have diagnostic value. The sound is *superficial*; seems to be near to the ear. The fact that it actually is produced near the surface of the chest makes it *amenable to pressure* with the stethoscope, its intensity being increased by gentle pressure, diminished or abolished by firm pressure. *Posture* also affects the loudness of a pericardial friction murmur, so that it becomes more distinct in the erect, and still more in the forward bending, posture; although in some cases, probably where the pericarditis is more apical and posterior, the reverse may be true. The effects of *respiration* are likewise inconstant, but usually the sound is intensified by a full inspiration whenever sufficient to cause firmer apposition of the rubbing membranes.

Broadly speaking, the intensity of pericarditic friction sounds depends upon (1) the properties of the exudate and (2) the strength of cardiac action. A fresh, soft, smooth fibrinous deposit, with feeble force of the heart, will give a hardly perceptible friction sound; on the other hand, a chronic, thick, hard, and rough deposit, with exaggerated cardiac force, will produce a loud and harsh murmur. Pericardial friction sounds vary in intensity from day to day, as well as in precise location. In patients with thin chest walls and a tumultuously pulsating dilated right ventricle, firm pressure with the stethoscope in the fourth or fifth left interspace may develop a rubbing sound which often closely simulates the pericardial friction.

It should be noted here that a variety of conditions besides simple fibrinous pericarditis may give rise to friction sounds. The principal of these are the following: Abnormal dryness or stickiness of the pericardial serous surfaces, as in cholera; milk spots on the visceral (right ventricle) pericardium; tuberculosis, carcinoma, and gummatous syphilis of the pericardium; calcifications and fibrous roughnesses from arterial sclerosis (Gibson).

DIFFERENTIAL DIAGNOSIS BETWEEN PERICARDIAL FRICTION SOUNDS AND ENDOCARDIAL MURMURS.—This is made by noting especially the differences in quality, in rhythm, in constancy of location and intensity, the variable, shifting position of the pericardial murmurs being in marked contrast to the constancy of endocardial valve murmurs. The superficial character of friction sounds, and their susceptibility to pressure of the stethoscope, are features. Other points are the temporary nature and irregular strength and time of the pericardial, and the chronicity and regularity of the endocardial murmurs.

Pleuropericardial Friction Sounds.—Murmurs may be caused by roughness of the outer surface of the pericardium, or of that portion of the pleura which overlaps the heart, or both. The resulting pleuropericardial friction sounds are similar in quality to the pericardial. They are most frequently heard in the fourth and fifth interspaces, where the lingula of the left lung is apposed to the apical portion of the heart. A distinguishing feature of these sounds, which are virtually pleuritic friction sounds in origin, is their behavior in relation to respiration. The friction is often heard only during inspiration, and during the cardiac systole; and while it may disappear when the patient holds his breath, it is only faintly audible, if at all, during the expiratory act. It is usually louder during a full inspiration than an ordinary one. I have seen a few cases in which the sound was heard as a pleuritic friction, with respiratory rhythm, during forced breathing, and as a pericardial friction during quiet breathing, with the rhythm of cardiac action alone distinct.

Subphrenic friction may be produced by a subdiaphragmatic peritonitis or a diaphragmatic (phrenic) pleuritis, the sound being synchronous with the heart action, and best audible at the lower end of the sternum and in the interchondral spaces.

Pericardial Splashing Sounds.—These, which may also have a churning quality, are heard if air and fluid be present in the pericardial sac, as from stab wounds. The condition is termed a *hydro-* or *pyopneumopericardium*, analogous to similar conditions of pneumothorax, the heart acting as a rhythmical succussion-splash producer. The sounds have been well likened, also, to those caused by a water-wheel. Obviously, they continue while the breath is held. Exceptionally, such sounds may indicate the disturbing effect of cardiac action upon adjacent large pulmonary cavities filled partially with gas and liquid exudate, or upon a dilated stomach. In the latter case, the sounds may have a metallic tone.

Cog-wheel respiration, in the majority of instances, is undoubtedly suggestive of a tubercular deposit in the bronchioles, and yet may occur as an accompaniment of a hypertrophied heart. In this exocardial phenomenon my experience tallies with the experience of Gibson. A gentleman, fifty years of age, of medium build but with muscles of iron-like resistance, was considered a good oarsman on the Schuylkill River when a young man. His lungs are entirely sound; nevertheless, he has, along with a strong, hypertrophied heart, interrupted inspiration over portions of his left upper lobe, anteriorly. It is probable that a pleuropericardial or cardiorespiratory murmur may be confused with cog-wheel respiratory sound of cardiac causation.

Crepitations, or fine crackling or moist râles, may be set up by and accompany the movements of the heart as other extrinsic manifestations. Thus, adjacent to and impinging upon the heart, the air-vesicles and smaller bronchi contain more or less viscid secretion; or there may be a mediastinal emphysema, in all of which these adventitious sounds occur in synchronism with the heart's movements.

CHAPTER XIV

EXAMINATION OF THE BLOOD-VESSELS

THE ARTERIES

INSPECTION

In connection with the physical signs of diseases of the heart, no rational, successful, or useful diagnosis can be made without a careful consideration of the vascular phenomena. The diagnostic value of the evidence to be obtained by observation of the cervical and peripheral vessels is a common fact in the interpretation of cardiac conditions. The pipes are organically, vitally, semeiologically related to the pump.

The Arch of the Aorta and the Larger Arteries.—Visible pulsation along the course of the aortic arch is usually significant of *aneurism*, especially if accompanied with swelling. If the pulsation is seen to the right of the sternum, the ascending portion is involved. When the manubrium or the suprasternal notch pulsates, the transverse portion of the arch is affected. It should not be forgotten, however, that not all aortic pulsations observed are necessarily abnormal; as in the episternal notch, it may be due to a high location of the arch, rarely to mere old age. On the other hand, the aortic pulsation may be due to retraction of the lung. Aneurismal pulsations are invariably systolic. Sometimes a pulsation in the second right interspace may mean dilation of the commencement of the aorta, associated with aortic insufficiency and marked hypertrophy of the left ventricle; but it usually has a more diffuse, wavy character instead of the finer, shorter, more circumscribed aneurismal impulse.

The **pulmonary artery** may be seen to pulsate in *retraction of the left lung* from fibroid phthisis or chronic pleural adhesions. In a case of cerebrospinal meningitis, in a boy of six years, I once witnessed the development of acute endocarditis as a complication, with decided pulsation over the pulmonary artery, palpable as well as visible, and accompanied with a systolic murmur heard best in the same region. Aneurismal pulsation of this artery may also occur, but it is rare.

The **innominate artery** may give rise to visible pulsation near the right sternoclavicular articulation, and even to some bulging owing to aneurism, or possibly to disturbance from involvement by aneurism of the aortic arch.

The **carotid arteries** show a slight movement of pulsation above the clavicles in many healthy persons. Decided visible pulsation of the carotids may indicate nervous excitement or emaciation without any cardiac abnormality.

Marked throbbing in the neck, however, is most typically caused by the combination of *cardiac hypertrophy* and *aortic insufficiency*; in fact, it is a common phenomenon in most instances of imperfect filling of the arterial system with relaxation or dilation of the vascular walls, as is met with in *anemia*, the *hypoplasia* of *chlorosis*, *exophthalmic goiter*, etc. *Arteriosclerosis* may also so affect these arteries as to produce visible pulsations, owing to the prominence of the thickened, rigid, tortuous walls.

Abnormal pulsations of the **other arteries** are due to the same causes that have just been enumerated. It is in *aortic regurgitation*, however, that the most distinctive pulsations of the principal arteries are noticeable. We find this especially true of the subclavian, axillary, brachial, and radial arteries; less so of the ulnar, femoral, anterior and posterior tibial, and dorsalis pedis arteries. The mode of production may be indicated as follows: The sharp, exaggerated rise of the pulse-wave is due to the hypertrophied left ventricle, and the inelastic vascular walls usually associated. As soon as cardiac diastole begins the intravascular pressure is suddenly released, aided by the regurgitating current of blood into the left ventricle, thus making the pulsation manifest by the extreme amplitude of rise and fall. Prominence of the temporal artery and of its pulsation is a common observation in those more or less old people who show the vascular changes of fibrous thickening. Not infrequently one may notice inequalities in the course of a sclerosed artery, due to the collection of lime salt deposits. Sometimes, also, a lateral movement of a prominent and tortuous brachial artery at the elbow, or of a temporal artery, may be observed as well as the vertical pulsation.

The Abdominal or Descending Aorta.—This is seldom seen to pulsate in the healthy individual, except temporarily from exertion and excitement. Reference has already been made to aortic pulsation in the epigastrium; its production by immediate disease, as aneurism, or by transmission through an overlying pyloric, pancreatic, hepatic, mesenteric, or other tumor. In distinct contrast to the circumscribed pulsation of an abdominal aortic aneurism is the diffuse pulsation—violent it may be—of a large portion of the vessel. This is characteristic of many patients who are neurotic, hysteric, or neurasthenic, and who suffer reflexly from various complaints, as chronic gastro-intestinal catarrh, movable kidney, ptosis of the abdominal organs, ovarian disorders, tuberculosis, peritonitis, and the like.

PALPATION

The Aorta.—This great vessel is not palpable normally, except in certain anomalous cases of high position of the arch, when its pulsations may be felt in the episternal notch by making deep pressure downward behind the sternum. In acute or chronic dilation (diffuse), with or without aortitis, the aorta may be felt in the right first and second interspaces, and if the transverse portion of the arch is involved, also over the manubrium as a transmitted impulse. It is in the diagnosis of *aneurism* that palpation has decided confirmatory value, noting the area, force, and expansile character of the pulsation, thrill, and diastolic shock. Here may be described also the palpatory sign of thoracic aneurism known as the "*tracheal tug.*" It is elicited by the physician standing behind the seated patient, whose head is bent well back and chin elevated. The examiner then inserts the tips of both index fingers under the lower edge of the cricoid cartilage, which is gently raised by them (Ewart), when, if aortic aneurism be present, an unmistakable downward tug is felt with each cardiac systole. Tracheal tugging occurs only when the aneurism is so situated and connected with the transverse portion of the arch of the aorta that it presses downward upon the trachea at its bifurcation.

The **pulmonary artery** or second left interspace region may be the seat of pulsations that may be palpated. Here, near the sternum, may sometimes be felt, an instant later than the apical impulse, a circumscribed pulsation or diastolic shock, due to a sharp recoil upon the pulmonary valve leaflets from heightened pressure in the pulmonary circulation, and is thus an indication of hypertrophy of the right ventricle, especially noticeable if there is simultaneous retraction of the anterior border of the left lung. Such marked hypertrophy of the right ventricle is usually the result of mitral stenosis.

The palpable signs of the other arteries and their pulsations may be included in the consideration of the traditionally and commonly and quite appropriately selected radial artery and pulse. As an index of the cardiac condition and of vasomotor tone, the anatomic situation and practical convenience of access of the radial artery make it sufficient and satisfactory, although on occasion the carotid and temporal arteries serve a useful purpose.

The Pulse

In these days of instruments and methods of precision in the examination of the heart, much less attention is being paid to the pulse in the clinical instruction of medical colleges than should be. In practise, however, it is doubtful whether the busy physician in private or hospital work fails to acquire quickly and comprehensively most important data which, although bearing upon the condition of the heart, could not be determined so promptly by an examination of that organ itself. This is especially true of emergencies, and in multitudes of instances where judgment and skill in diagnosis, prognosis, and treatment are urgent factors. Hence it is that training in taking the pulse can never be too full, constant, and diligent, so that the most exquisite sensitiveness of touch may be developed in conjunction with related clinical and pathologic knowledge—feeling with the brain as well as with the fingers. There are several instrumental auxiliaries in ascertaining the character of the pulse, such as the *sphygmograph*, for making graphic pulse tracings, and the *sphygmomanometer* and *tonometer*, for determining the blood-pressure; and while both are scientifically and practically valuable in diagnosis, they can never take the place of the convenient, well-trained, sentient finger-tips.

Method of Examining the Pulse.—The *position* of the patient's body is important, especially as it influences the frequency of the pulse. All muscular constraint must be forestalled by putting him at ease in a reclining or sitting posture. The arm whose radial pulse is to be palpated should be allowed to rest upon the side of the bed or couch, a table, or chair support, or should be held by the examiner's other hand. If the patient's left radial artery is to be examined, the observer stands or sits to the left, and employs his left index, middle, and ring fingers, or, as I often prefer, the first two of these, with their tips just inside and above the styloid process of the radius, the patient's wrist being held in a semiprone position. For the right artery, the physician palpates similarly with the right hand while at the patient's right side. Exceptional conditions of convenience, necessity, adaptability, and the personal equation may alter or reverse this method. When, as should always be done, both radials are examined simultaneously, the observer stands in front of the patient, using his right hand for the right artery, and his left for the left. The technic here given affords the accomplishment of placing the index finger proximally—that is, nearest to the heart—and thus enables this most sensitive finger to regulate the pressure upon the artery, the effects of which may then be appreciated by the other fingers (Gibson). The palpating pressure should at first be light. Not only should both arteries be palpated, at least on the first examination, for the estimation of any possible asymmetries of pulsation—to be referred to later—but also to discover any anomalous position. For it is not uncommon to find the radial artery winding around the upper part of the styloid process of the radius to the back of the hand, leaving the bony base of the artery in front without pulsation, or with a slight, deceptive pulsation, produced probably by the superficial volar artery.

Other points in the technic will be indicated as the conditions to be ascertained specifically are described seriatim.

Phenomena to be Observed.—It is well to adopt a habitual order of procedure in studying the details of the artery and pulse. Thus we may gain a threefold impression, mainly: (1) The *condition of the artery*—its walls; (2) the *condition of its blood-supply*—its fulness; (3) the *character of the pulse-wave*—its pulsatory dilation and contraction.

In palpating the pulse, it will be noticed that we determine three important factors intimately connected with heart affections: The elasticity of the arterial system is inferred; the cardiac action, and the resistance in the arteries and capillaries, each of these two latter factors influencing both the second and third impressions indicated above.

(1) THE ARTERIAL WALLS. *Technic.*—By digital pressure a little above the palpating region, the radial artery may be emptied, and rolled under the other fingers by slipping the skin over it to and fro, at the same time pressing the vessel gently against the bone. In this way the size or caliber of the artery, and the condition of its walls, may be felt. The *normal size* of the radial arteries may vary decidedly in different individuals, and its determination must be largely a matter of judgment based upon many observations, and upon considerations involved in the build of each person. The estimation must be made from the sensations derived between the pulsations

order to be truly representative of the actual arterial caliber. One may note small arteries in a large, stout man, or large arteries in a lean man, exceptionally, but usually we find large-sized arteries in individuals of large build and large hearts, and smaller ones in those of small musculature and stature.

Pathologically we seldom meet with a radial artery which can be denominated as oversized. It may, however, be found in certain persons of obese habit, inclined to pallor rather than to plethora (the "lymphatic temperament" of the older writers), and with a notable flabbiness of the walls as an associated sign. This is frequently an indication of accompanying cardiac weakness or lack of myocardial tone and vigor from hypoplasia. On the other hand, abnormally small arteries are more commonly met with, often as *congenital* conditions, when they may be mistaken for weak, small, thready pulse-waves, which, of course, do occur with them, for obvious anatomic reasons, and not because of diminished heart power. Hence, as Cabot points out, it is important to distinguish such a *small pulse-wave* from a really *weak pulse.* The *wiry, contracted artery* of chronic lead-poisoning, etc., may more readily be palpated between the pulse-beats because of its resistant qualities.

The *normal* physical condition of the arterial walls gives the palpating finger the feel of resiliency or elasticity with slight or moderate pressure. It should be remarked, however, that in running the finger along the artery it is difficult to feel between pulsations except in a very thin wrist, because of the fact that the normal walls are soft and yielding as well as elastic.

The principal *abnormality* is due to atheromatous degeneration and thickening, which give rise to tortuosity, hardness, rigidity, and varicosity of the walls. The discovery of one or more of these signs in the radial artery is confirmatory of a widely distributed *arteriosclerosis* of the peripheral arteries, and usually of some hypertrophy of the left ventricle. The radial may simply feel unduly firm and stiff, with little or no tortuosity, as a result of fibrous thickening, and may thus be rolled under the fingers like a cord. Sometimes several inches of the artery may be traced up the forearm, the pulse-wave passing under the fingers as a distinct worm-like movement. When the artery feels varicose or "beaded," as if girded at short intervals with hard rings, *calcification,* or the deposit of lime salts, is present. Strictly, the varicosities are due to the bellyings between the calcified constrictions. In aggravated cases the calcification is so diffuse and the hardening so continuous as to give rise to the well-known term, "pipe-stem arteries." Here the pulse may be practically obliterated, so as to cause the artery to simulate a tendon. Hardening of the arteries must not be confounded with increased tension of pulse (see later).

(2) ARTERIAL VOLUME, OR THE BLOOD-SUPPLY.—This may be estimated by palpating the artery at its systole—that is, between the pulse-beats—as in the case of the vessel itself. Indeed, the sense of fulness which the radial may possess is very like that of its caliber, with this difference, that whether the artery be large or small, its sense of fulness is accompanied by a greater resistance or tension than otherwise. A full artery (*arteria plena*) is to be discriminated from a full pulse, and a comparatively empty artery (*arteria vacua*) from an empty pulse; as a high-tension pulse, with small wave amplitude, may yet occur in a full artery, and a full pulse is often felt in certain wasting diseases where the actual blood-supply is markedly deficient.

The *pathologic states*, then, which give rise to these two types of arterial fulness are three: (*a*) abnormal *quantity* of blood, either *plethora* or *anemia*; (*b*) degree of *cardiac power*, strong or weak; (*c*) *resistance* in the arterioles, great or slight, arteriosclerosis and chronic interstitial nephritis being examples of the first, especially in the early stages, the relaxation of malnutrition, chronic wasting diseases—as cancer—some acute febrile diseases, and aortic incompetency, examples of the second.

(3) CHARACTER OF THE PULSE.—Ordinarily the pulse-waves are studied first (and these counted per unit of time), the preceding points being left until later. There should be noted: (*a*) The *rate* or *frequency*; (*b*) the *rhythm* or *regularity*; (*c*) the *quality* or *character*; (*d*) the *symmetry*.

The *normal pulse*, obviously, has all of these elements, individually correctly developed and collectively relatively balanced. In short, the pulsations should, to be normal, have a rate variable only within certain limits—a regular rhythm—and a moderate and uniform volume, force, form, duration, and tension or sustained pressure.

(*a*) *The Rate of the Pulse.*—The *normal pulse-rate* varies within certain limits, and according to certain influences to be mentioned shortly. The average frequency in the adult male, however, is 72 per minute; in the female, about 74, although many women have a normal rate of 78 or 80 pulse-beats per minute. The personal idiosyncrasies ordinarily met with cause a pulse-rate seldom under 60 or over 80 in those of middle life. In the youngest infants the pulse frequency is normal at about 140 beats to the minute; when one year of age, 120 to 130; at three years, 100; at the fifth and sixth years, from 90 to 95. The normal pulse is quite susceptible to acceleration from exertion, excitement, anxiety, and other emotional states, after meals, during the later hours of the day, in hot weather, in the erect attitude, after forced breathing, etc. The pulse-rate may be normally slow as a congenital condition, in old age, and immediately after childbirth.

It is not within the province of this book to enumerate even the multitude of pathologic conditions of which increased or decreased frequency of the pulse is symptomatic: the fevers, inflammations, intoxications, neuroses, degenerations, and various functional and organic affections of the cerebrospinal system, thorax, abdomen, and so on. It will suffice to point out briefly the cardiac disorders which cause abnormal pulse-rates.

(i) Increased Frequency (*Pulsus frequens*).—The pulse may be increased in frequency in all forms of valvular disease except in aortic stenosis; in cardiac weakness or dilation (heart-failure), from whatever cause; in the cardiac neuroses, such as nervous palpitation from tobacco, tea, alcohol, and venereal excesses, exophthalmic goiter; and in paralysis of the vagus or irritation of the sympathetic nerves or of the intracardiac ganglia. The various forms of simple increase of rapidity of the heart, with corresponding pulse-rate, are included under the term *tachycardia.* Extremely rapid pulse is a characteristic of those more essential, sudden, alarming attacks known as a *paroxysmal tachycardia,* lasting from a few minutes to several days. The pulse-rate may go as high as 200 per minute, or even more. This affection may be due to coronary-artery disease, or to areas of necrosis in the myocardium. Similarly, in angina pectoris there is decided increase in pulse-rate frequency. The pulse also reflects the cardiac condition as affected by various thoracic and abdominal diseases, by anemia, tuberculosis, rheumatoid arthritis, etc. In the chronic, apyrexial diseases of the heart, where the valvular and myocardial lesions predominate, the rate of the pulse varies from 80 to 120 beats per minute.

(ii) Decreased Frequency (*Pulsus rarus*).—Abnormally slow pulse is the direct result of slow heart—*bradycardia* or *brachycardia*—or a rate usually below 60. It should be remembered that the pulse may be normally infrequent as a personal idiosyncrasy, or even a family characteristic. A rate of 40 or under is generally considered of very grave significance. In an elderly woman with cardiac dilation due to mitral insufficiency, a fatal attack of influenza supervening, I noted a pulse-rate of 26 beats per minute. In fact, during the prevalence of severe influenzal epidemics in the last decade of the nineteenth century, bradycardia was a frequent complication and temporary sequel.

Among the other maladies which are chiefly responsible for the bradycardial origin of slow pulse, the following may be mentioned: (a) Cardiac disease, such as affects principally the myocardium—fatty degeneration, especially when due to atheroma of the coronary arteries; chronic fibroid myocarditis, and sclerotic stenosis of the aortic orifice. (b) Increased arterial tension and resistance from poisonous substances circulating in the blood and affecting the cardiac centers or ganglia, as in auto-intoxication from chronic digestive disorders, jaundice of hepatic origin, chronic Bright's disease (chronic uremia); also from chronic lead, alcohol, and illuminating-gas poisoning. (c) Cerebral and meningeal diseases which increase the intracranial pressure, and consequent irritation of the vagus, as tumors, effusions, hemorrhages; or there may be peripheral pressure upon this nerve or the cervical cord from tumors, injuries, and the like. (d) *Essential bradycardia,* a persistent condition for years, supposed to be without any definite lesion, but often really due to sclerosis of the coronary arteries, or perhaps of the medullar arteries about the vagus center. When associated with epileptiform attacks the condition is known as the Stokes-Adams syndrome. (e) Myxedema, melancholia, the convalescent stage of the acute infectious, febrile diseases, asthmatic paroxysms, and inanition may all cause infrequent pulse. Care should be exercised not to mistake an intermittent for a slow pulse because of the untransmitted pulsations. This error may be avoided by auscultating the heart simultaneously with radial palpation.

(*b*) *Rhythm of the Pulse.*—The normal pulse-waves succeed each other with complete regularity in respect to time of contact with the finger and of interval between the waves; also in respect to force and other qualities pertaining to the successive beats. Again, however, we meet with individuals occasionally whose pulse-beats manifest slight irregularities of rhythm, but in whom not the least evidence of cardiac or reflex origin is discoverable. Nevertheless, it may be found some time that these apparent idiosyncrasies may prove to be congenital, autotoxic, or neurotic in causation. Physiologic deviations may occur in old age, after severe exertion, and during intense mental excitement. I have observed in several persons a slight acceleration near the end or just after the height of the inspiratory act for two or three beats, the rate subsiding during the pause between the respirations—a sort of regular irregularity.

Arhythmia signifies virtually a disturbance of the nervous mechanism governing the automatic contractions of the heart. The causes of such deranged innervation may be grouped into two classes: those which produce *intermittency* of the pulse and those which produce specific *irregularity.*

Intermittent pulse (*pulsus intermittens,* or *deficiens*) means one dropped beat at certain intervals, which may be regular or irregular. That is, the heart may skip a beat after two contractions (*pulsus bigeminus*), or after three (*pulsus trigeminus*), in regular sequence; or the intervals of pulsations may range from three to twenty or more —seven, then four, then ten, and so on irregularly with the intermissions. When the intermission is due to a real pause in the cardiac action, it is termed a *deficiens* pulse; when due simply to an occasional weak contraction of the heart (determined by auscultation), as an *intermittens* pulse. Closely related to the deficient and intermittent, the bigeminal and trigeminal pulses, and, as Vierordt says, standing somewhat between these and complete irregularity, is the intercalated or *intercurrent* pulse (*pulsus intercidens*). It is an appended instead of a suspended pulsation, suddenly and immediately following two or more regular pulsations, the last one of which may be slightly weaker than the other ones, and itself followed by a slight but distinct pause. This type of pulse is usually the evidence or premonition of serious myocardial weakness.

Suspended or intermittent pulse is not characteristic of any disease or condition, and may be present without any subjective knowledge by the patient as affecting his health. It may be habitual, a constitutional peculiarity, or the manifestation of a neurosis, or of coffee, tea, tobacco, or digitalis intoxication. It may disappear during fever or temporary acceleration of the heart from various causes. Practically it is never as serious a sign as true irregularity, and yet does accompany, sometimes, such organic conditions as valvular disease, especially mitral, and chronic myocarditis, with the patient at rest, but is apt to turn to genuine irregularity with the patient about and under the stress of broken compensation.

Irregular pulse (*pulsus inequalis*), here dealt with as to time or as a true arhythmia, pertains also to quality, including volume and force or pressure of pulse-wave, to be described shortly. It is characterized by irregularity of *interval* between the succession of pulsations, and of the *rate* or rapidity of their succession. That is, the time between some of the pulse-beats differs and varies in length from others, or they may follow each other in fast, and again in slow time.

Although a pulse of irregular time may occur in some elderly persons of apparently good health, in infancy and at puberty, after overexertion or dissipation, in the majority of cases it probably means myocardial degeneration, with or without mitral (especially stenosis) or aortic disease, sclerosis of the coronary arteries, or some grave involvement of the nervous cardiac mechanism from toxemia—diphtheric, alcoholic, theic, diabetic, therapeutic (digitalis), uremic, etc. In the variety of arhythmia known as *tremor* or *delirium cordis*, a part of the irregularity is due to a paroxysmal, rapid fluttering action of the heart reflected in the pulse action, and may be found associated with syncope, gastro-intestinal disorders in which flatulence is an aggravating symptom, and with the later stages of fatal cardiac conditions, especially the myocarditic in gouty, obese, and atheromatous subjects.

(*c*) The *quality of the pulse* really embraces the most important and diagnostically significant of the phenomena to be palpated. Thus we study (i) the *force* or *compressibility*; (ii) the *volume* or *size*; (iii) the *tension* or *elasticity*; (iv) the *shape* or *form* of the pulse-wave; (v) the *equality* or *regularity* of the pulsations as to these attributes.

(i) The *force* of a pulsation is an index of heart strength, as tension (iii) is of arterio-capillary resistance. It is determined by the degree of finger pressure required to obliterate the pulse-wave. The technic is as follows: Palpating with two fingers, the index and middle, while pressing moderately and steadily with the proximal finger (nearer the heart), with the other (distal) the force or compressibility is estimated. If with such pressure the pulse-waves are obliterated, the pulse is spoken of as weak or compressible; if distinctly felt, as strong or incompressible.

Allowance must be made in those having wrists well padded with fat, as the pulse strength may be quite normal in fact, although more easily obliterated by moderate external pressure.

Abnormal *increase* in the force of the pulsations of the radial, which may be temporary or chronic, indicates exaggerated ventricular systole, and therefore the muscular power and activity of the heart—usually an hypertrophy.

On the other hand, muscular cardiac debility is indicated by a *compressible* pulse, as is found in fatty degeneration and dilation.

As the actual strength of the pulse-wave is closely related to and conditioned by the arterial tension, practically the estimation of the force or compressibility must culminate in a balanced judgment upon a careful consideration of the two factors—cardiac and vascular—in combination. Of course, the greatest degree of intraradial pressure will be noted where hypertrophy of the left ventricle and intrinsic hypertension of the arterial walls exist together. The force of the pulsations is but slightly dependent upon the amount of blood in the arteries.

A difference in the strength of the radial pulse as the arms are raised and lowered, comparing one side with the other, as pointed out by Sorgo, may be indicative of traction upon the subclavian artery by the pathologic apex of a tuberculous lung.

(ii) The *size* of a pulse-wave (*volume* of the pulse) also depends upon the two factors mentioned just before, namely, the force of the myocardial systole and the tension of the arterial walls. The amplitude of each pulse-wave is estimated by the amount of diametric expansion palpated: this may be large, medium, or small.

Larger pulse (*pulsus magnus*) than normal may be associated with plethora, hypertrophy of the left ventricle, and relaxed conditions of the arterial walls. The large pulse is observed best in cases of cardiac hypertrophy accompanied with soft, large, weak walls of the vessels. This may often be found in cases of *aortic incompetency*, where the left ventricle is usually enormously enlarged and the arterial walls flabby and relaxed. Two exceptions to the influence of cardiac hypertrophy upon the size of the pulse-waves should be noted. In *mitral regurgitation* the pulse may be medium or small in volume, notwithstanding the hypertrophy, because with each systole part of the blood in the left ventricle leaks back into the left auricle. Again, in *aortic stenosis*, the narrowed orifice prevents even the hypertrophied ventricle from forcing the normal, full volume into the aorta. Abnormally large pulse is also observed in many febrile conditions (*bounding* pulse); temporarily from the debilitating effects of hot, humid weather, and in some persons affected with obesity, tuberculosis, etc., before decided cardiac weakness has supervened.

Abnormally *small pulse* (*pulsus parvus*) occurs in cases of cardiac weakness and constriction of the arteries. By contrast with large pulse, this has already been referred to in connection with aortic stenosis and mitral regurgitation. In *mitral stenosis* we have a small pulse because of inability of a sufficient quantity of blood entering the left ventricle to be propelled into the aorta, and the weak, atrophic condition of the ventricle itself, due to this very fact of lack of stimulus and work, and the consequent partial disuse. A small or "thready" pulse may be associated with *aneurism* of the aorta, with *chronic Bright's disease* and *acute peritonitis*, and in *inanition*. It is usually an accompanying phenomenon, also, of the form of arhythmia, described before, known as the trembling or delirious pulse (*pulsus tremulus*).

The size of the pulse-wave has no necessary relation to the force or intravascular pressure. A strong pulse is not necessarily a large one, as the arteries may be contracted and resistant; nor is a weak pulse necessarily a small one, as a full, large pulse may be associated with marked feebleness of cardiovascular tone.

(iii) *Tension.*—By this term is meant the feeling of pressure communicated to the fingers by the blood through the arterial walls. It includes the estimation of the pressure between the beats as well as the pulsations themselves—that is, the diastolic as well as the maximum or systolic pressures (corresponding to the systole and diastole of the arteries, respectively). Practically, this character of the pulse is observed in the same manner as the force of the heart action is judged—by noting the degree of compressibility. Therefore, we speak of two deviations from the normal tension or tightness due to intravascular pressure: the *hard pulse* (*pulsus durus*) and the *soft pulse* (*pulsus mollis*).

In estimating the tension, care must be exercised in discriminating it from either the sclerotic hardening of the arterial walls, on the one hand, or the fatty atheromatous softening, on the other; the arterial walls must be considered apart from the actual blood pressure from within, and its independent effects upon them. Considerable practise is necessary before skill is attained in properly and efficiently gaging the pulse-tension. For, although instrumental aids are available for scientific and, occasionally, hospital work, in 99 out of 100 cases the fingers must remain the most convenient, sentient, and practically trustworthy agents.

Instruments.—Right here, the apparatus used as diagnostic aids in studying the blood pressure may be referred to briefly, the student obtaining further information and details in works on physiology and general medical diagnosis.

The Sphygmomanometer.—Blood pressure instruments have been constructed along two different lines: one aiming to eliminate the subjective factor by a simple instrument accurately and graphically recording the blood pressure, the other to obtain the systolic or maximum blood pressure simply by exerting sufficient instrumental pressure to obliterate the distal pulsations. In the latter method, v. Basch invented the first instrument of any value, although not scientifically accurate, and yet useful, perhaps, in aiding along lines of indications and results of treatment (C. E. Brush, Johns Hopkins). The former method is exemplified in the instrument of Roy and Adami, in 1890.

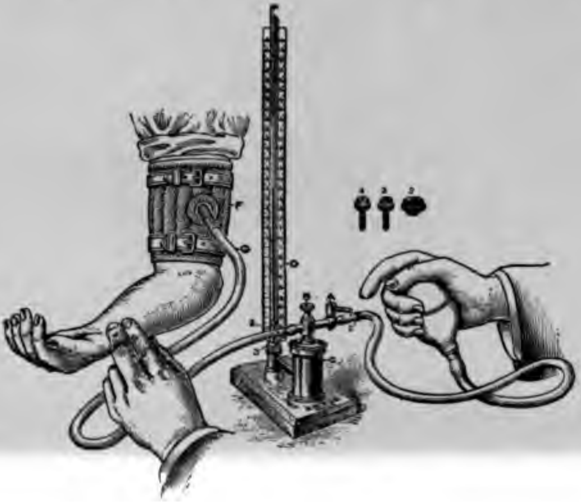


FIG. 68.—STANTON'S SPHYGMOMANOMETER.
(Cut from The Arthur H. Thomas Co.)

Of the v. Basch type, the most commonly used sphygmomanometer is the Riva-Rocci. It consists in the application to the middle of the upper arm of a cuff like a pneumatic tire, about 12 cm. wide (as improved). This is connected with a mercury manometer by a piece of rubber tubing, to which is also joined similarly a rubber hand bulb, thus forming a closed system. "The pressure is raised by compressing the bulb until the radial pulse can no longer be felt, when the height of the column of mercury in the manometer will represent the pressure necessary to overcome the pressure of the blood in the artery, or, in other words, the blood pressure" (Musser).

By the methods of Erlanger and Strassburger, more scientific and satisfactory results may be obtained, in that both the systolic and diastolic pressures are graphically recorded. The cumbersomeness and expensiveness of these instruments are the principal objections to their use by the general practitioner.

Gaertner's *tonometer* is another variety of apparatus for estimating the sustained pressure within the arteries. Although more convenient to use, it is inaccurate, because dependent upon too many inadvertencies of technic, and the uncertainties of the subjective criterions of both patient and physician. In any event, both systolic and diastolic pressures should be taken, the difference between the two representing the real blood pressure.

The Normal Blood Pressure.—This varies according to the influence of a number of physiologic factors, such as bodily position, age, sex, rest, exercise, excitement, the taking of food, the time of day, etc. The tension is higher in the erect than the sitting position, and in the sitting than in the decubital position. Gennari found, however, that immediately after rising from a reclining posture, in health, there was a slight drop in the blood pressure. In fatigued and temporarily debilitated conditions, this fall of pressure on rising is still more noticeable, being marked just before syncope. The tension in adults is greater than in children, and still more marked in the aged. It is usually a trifle less in women than in men, greater after physical, mental, social, and emotional exertion and excitement, a full meal, and during the most strenuous working hours of the day. Generally, the *average normal blood pressure* may be stated as about 120 mm. of mercury in the manometer. Below 110 mm. and above 160 mm. Hg are considered abnormal pressures, the comparison being made with the subject at rest.

Or Strassburger's "blood-pressure quotient" may be adopted to express the condition. This is obtained by dividing the blood pressure—the difference between the systolic and diastolic pressure—by the systolic pressure. Normally, this averages 0.25, and its variations aid in determining whether the changes are due to stronger or weaker ventricular contractions, or to increased or decreased peripheral resistance.

Pathologic Blood Pressures.—The high-tension or hard pulse occurs when one or both of the two principal factors are operating—increased peripheral resistance, especially, and increased cardiac force. The pressure of a large volume of blood, as in plethora, must also be considered in many cases. Whatever the singleness or combination of these causative conditions, the pressure is at its maximum during the cardiac systole. Nevertheless, it is the pressure or tension of the pulse between the beats, the prolonged and sustained blood pressure and hard pulse, which is of most value in diagnosis. For there may be a high-tension pulse which is accompanied by a low pressure between the beats, as in aortic insufficiency, thus indicating strong cardiac action, but not increased peripheral resistance. Indeed, the discovery of a high-tension pulse is of direct importance in the diagnosis of the latter condition as its chief cause.

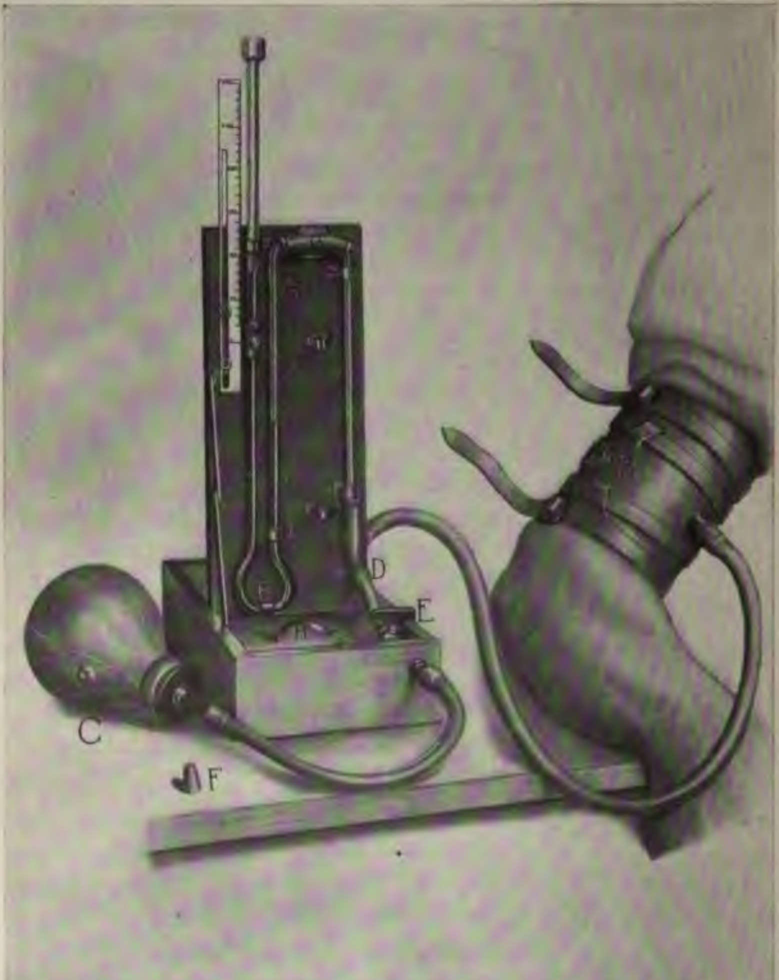


Fig. 69.—Janeway's Sphygmomanometer.

High blood-pressure pulse is characteristic in chronic Bright's disease and arteriosclerosis with contracted kidney; in gout and chronic autotoxemia leading to arterial atheroma; in chronic lead-poisoning; in cerebral hemorrhage (often very high); diabetes in elderly, gouty persons; chronic emphysema; angina pectoris; hypertrophy of the left ventricle.

A high-tension pulse is at the same time frequently a small pulse, and one in which the wave is of long duration, comparatively, in passing under the palpating finger. As the great value of diagnosticating hypertension in the arteries hinges upon its application to prognosis, prophylaxis, and treatment, practise in detecting it in its incipiency is a *sine qua non* if the permanent, incurable thickenings of the arterial walls are to be anticipated and avoided.

The *low-tension or soft pulse* requires but slight pressure from without to obliterate it. It feels as collapsible as if the artery were filled with gas. Because of the relaxed, non-resistant state of the vascular walls, the pulse-waves are usually high from free expansion with each ventricular systole. But if the heart is simultaneously weak and the dynamic quantity of blood in the arteries small, the latter may seem small and contracted, and yet manifest a low-tension or "running" or "gaseous" pulse, so that the artery between the beats becomes practically impalpable.

Dicrotism is a characteristic feature of the soft, compressible pulse, wherein the main, distinct wave is immediately followed by a slight secondary recoil wave. This is a common sign in typhoid fever, some cases of neurasthenia, anemia, and other forms of marked debility, and in aortic regurgitation, owing to the sudden fall of blood pressure as soon as the ventricular contraction ceases. The dicrotic pulse may not be detected in extreme rapidity of the heart action.

The pulse of low tension may be an accompaniment of many of the cardiac affections where myocardial incompetency ensues. It may be found in some persons in hot weather, and in others as a hereditary peculiarity. Gennari's experience leads him to believe that the fact that as in heart-disease the blood pressure varies very little with the patient's change of position, neuroses of this organ may be differentiated, because in his cases the pressure became much reduced when the patient sat up, and the pulse dicrotic.

Pulsus Paradoxus.—This is a variety of abnormal pulse in which the tension and other qualities are conversely affected by the respiratory function. Instead of the pulse tension being heightened by inspiration, as in the normal, in the paradoxical pulse the tension is low at the end of inspiration, and likewise high at the end of expiration instead of low in the normal at this period. At the same time, a converse relation of the volume of the pulse to the respiration is observed; that is, the pulsus paradoxus is characterized by decreased volume during inspiration, and increased during expiration; indeed, during a full and deep inspiration the pulse may be so small as to be hardly felt at all. This pulse is usually the result of some mechanical obstacle (pulmonary traction?) to the emptying of the left ventricle into the aorta during the inspiratory act. As such it is an important sign of *mediastinal pericarditis* with fibroid adhesions, in which the cords or bands pass from the sternal region in such a manner as to bend and constrict the great vessels near their roots when put upon the stretch by the inspiratory dilation of the thorax; venous engorgement and reduplicated second sound are usually associated.

Secondly, the *pulsus paradoxus* may be caused by a condition which increases the negative inspiratory pressure "from changes which prevent the free entrance of air into the lungs." Thus, it has been noted in *stenosis* of the *larger air-passages*, and is the more marked as there is accompanying feebleness of contractile heart power. It may also occur in simple pericardial effusion, adhesive pericarditis, and in some instances of large pleural effusion.

As systolic retraction of the lower part of the precordium may accompany adherent pericardium, and as the degree of this sign depends largely upon the strength of the heart, we usually have an index of the latter in the relative prominence of the systolic retraction and the paradoxical pulse, in inverse proportion; that is, the more marked the systolic retraction (and hence the stronger the heart) the less marked the pulsus paradoxus.

(iv) The *shape* of the pulse-wave may be judged by the palpating finger; thus, whether it be short and sharp, or *quick* in the sense that each individual wave contact has a brief duration, a sudden, quick rise and fall, or whether the wave has a slow, gradual, prolonged rise and fall. The latter, or *slow* pulse, is the *pulsus tardus*; the former—quick pulse—the *pulsus celer*. Again, to avoid misunderstanding and confusion, these accepted terms are to be distinguished from those indicating an exaggerated or diminished number of pulse beats per unit of time, namely, the terms frequent, infrequent, rapid, and so on; whereas here, quick and slow pertain exclusively to the duration and character of impact of the individual waves, and their manner of ascent and descent.

Sphygmography.—For purposes of accuracy, study, recording, and reference, the form and succession of the pulsations may be represented graphically by means of an instrument—the *sphygmograph*—well known to students in the physiologic laboratory. The sphygmograph records not only the shape of the pulse-curve for comparison with other tracings and with the palpatory signs, but affords an idea of other elements. It gives a clear exhibition of the arhythmic pulse, and of inequalities of volume, force, and tension. It depicts smaller waves or oscillations that ordinarily are too fine to be noticeable to the palpating finger, although it is true that these may have little or no decided diagnostic value. Doubtless, however, were sphygmographic tracings taken oftener, and studied generally with the assiduity given in the interpretation of Röntgenograms, the skill thus developed might find graphic meanings of accuracy and larger usefulness.

There are almost as many forms of the sphygmograph as of the stethoscope, but the ones in common use are practically improvements upon that devised by Marey, such as Landois's, Sommerbrodt's, Mahomed's, Richardson's, Dudgeon's, the last named being one of the best. Although of but limited and merely supplemental value in actual diagnosis, the sphygmograph is so well known to the average student and physician that a description of the mechanism and the technic of its application is omitted here, attention being directed merely to the understanding and significance of the tracings.

Normal Pulse Tracing.—The interpretation of the *normal sphygmogram* is comparatively easy when one notices that there are but two main trends to the curve representing each individual pulse-wave, namely, the sudden, nearly vertical, uninterrupted *stroke of ascent*, indicating the *rise of pressure*, and the gradual, oblique, undulating *stroke of descent*—the line of *falling pressure*. The former line is known as the *percussion-wave*, and ends at the apex curve. It corresponds to the sudden distending wave of blood pressure set up by the left ventricular contraction which drives the blood into the aorta. The descending limb represents a quick initial fall of pressure, exaggerated somewhat by the fall of the recording lever of

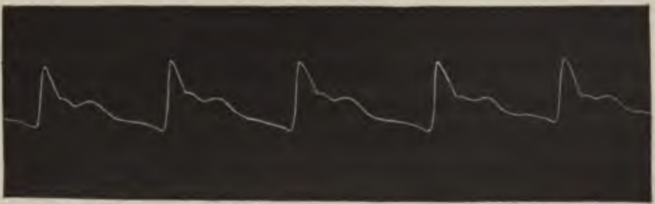


Fig. 70.—Tracing from the Pulse of a Normal Man. Pressure, 2½ oz. (Gibson and Russell.)

the instrument, which has been thrown too high by the distending impulse, followed by the *predicrotic* or *tidal wave*, which is believed to be caused by the fullest flow of blood, or, as by Roy and Adami, the completion of the intraventricular pressure ("outflow-remainder wave"). A second wave of interruption in the descending limb then follows, known as the *dicrotic* or *recoil wave*. This represents the reflected wave, probably from the periphery, due to the sudden checking of the column of blood against the aortic valve and the slight increase of pressure in the elastic arterial tube at the moment of oscillation. Occasional very slight oscillation waves may be noticed below the dicrotic wave, according to the degree of elasticity of the radial walls. It will be observed that in the normal tracing the apex is moderately acute, the tidal wave very small, and the dicrotic wave distinctly marked.

Pathologic Pulse Tracings.—A comparative study of the abnormalities of sphygmograms is readily made by noting the deviations from the normal in the details of the curve, taken in systematic order. It is here that the shape or form of the pulse-wave, as delineated by the sphygmograph, serves as a *diagnostic aid* to palpation.

We consider, therefore, the meaning of variations of the percussion-wave or upstroke, its height and obliquity; the breadth or acuteness of the apex; the prominence or absence of the predicrotic and dicrotic waves; the general character of the descending line, and the regularity of the base line.

a. *The Upstroke.*—*Increased height* indicates large volume, low tension, and a sharp systolic contraction of the heart; hence it occurs in aortic regurgitation (*pulsus celer*), and in the early stages of febrile states, where the arterioles are considerably relaxed.

A *short upstroke* points to slowness rather than suddenness of systole, heightened tension, and diminished amplitude of pulse-wave, and thus is found in such conditions as aortic stenosis, contracted kidney, gout, mitral regurgitation (moderate tension).

Obliquity of the ascending or anacrotic limb is practically nil in the normal sphygmogram. A marked *sloping backward* may be due simply to a weak ventricle, or may signify aortic obstruction, aneurism, or to a thick layer of subcutaneous fat over the radial artery.

On the other hand, when the tracing shows an actual *bending forward* of the line of ascent, it means a quick systole, whereby a large volume of blood is shot into the aorta suddenly, with low tension, and either a strong or weak heart. This is observed again in aortic incompetency.

b. *The Apex.—Increased breadth* of the top of the percussion-wave indicates a slow, forcible filling of the artery; a strong, hypertrophied heart laboriously contending against increased peripheral resistance or aortic obstruction, or aneurism, arteriosclerosis, etc. Consequently the tension is also usually prolonged.

Acuteness of the apex goes with the signs of a soft peripheral circulation (low tension) and quick initiative of ventricular contraction; therefore, with high and vertical ascending line, as in aortic insufficiency.

c. *The Tidal and Dicrotic Waves.*—A *marked tidal wave* (*pulsus bisferiens*) points to the conditions of high tension—hypertrophied heart, and aortic stenosis or thickened arteries. At the same time the apex of the tracing is low and blunt.

Small or absent predicrotic wave indicates a weak heart, or a moderate or low-tension pulse with a heart of fair or good strength, as in mitral insufficiency.

A *marked dicrotic wave* is usually, although not always, a sign of low-tension pulse with strong heart. When the notch preceding the wave of recoil reaches the base line it is said to be *fully dicrotic*; when it falls below this line it is *hyperdicrotic.* Dicrotism is likely to be marked in the later stages of depressing fevers, in tuberculosis, and the like.

Diminished or suppressed dicrotic wave signifies persistent high (prolonged) tension and forcible left ventricle, preventing the recoil. It may be a negative feature of the sphygmogram of aortic regurgitation, because the aortic check to the column of blood at the beginning of cardiac diastole is more or less removed.

d. *The Descending Line.*—There may be a general obliteration of the normal characteristics of this line, the slope, length, waves, and notches being totally altered, and irregular throughout. One finds these changes frequently in the sphygmographic tracings of aortic and mitral stenosis, in the later stages of mitral regurgitation, and in atheromatous thickening of the arteries.

e. *The Base Line.*—This, itself, may manifest irregularities and undulations, especially where cardiac disease is accompanied with marked dyspnea or Cheyne-Stokes breathing. Thus it may be seen in mitral obstruction, in chronic Bright's disease with dilated heart, in tuberculous meningitis and pericarditis, and in certain instances of marked dicrotism.

(v) *Inequality* of the pulse-waves (*pulsus inequalis*) or irregularity exclusive of the element of time simply refers to a complexus

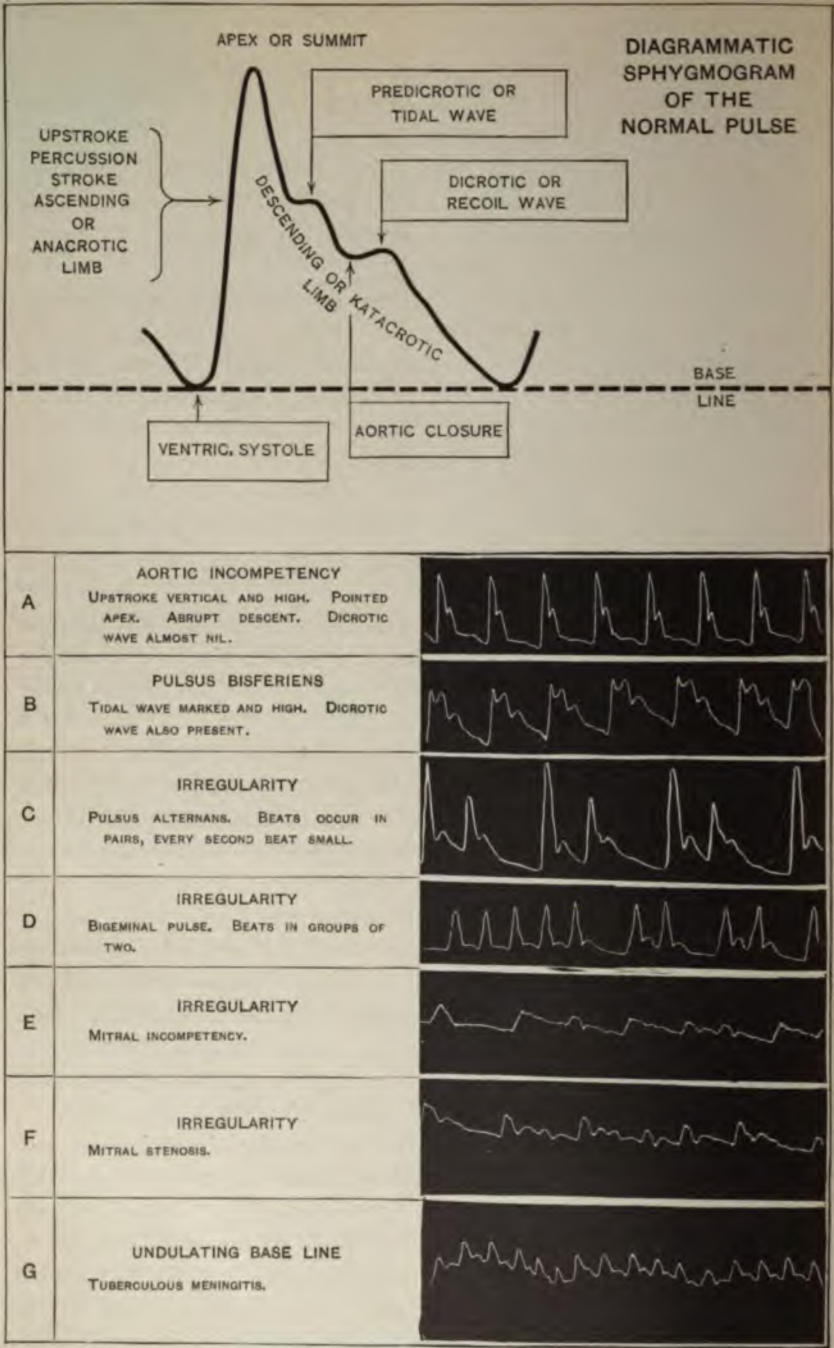

APEX OR SUMMIT
DIAGRAMMATIC SPHYGMOGRAM OF THE NORMAL PULSE
PREDICROTIC OR TIDAL WAVE
UPSTROKE PERCUSSION STROKE ASCENDING OR ANACROTIC LIMB
DICROTIC OR RECOIL WAVE
DESCENDING OR KATACROTIC LIMB
BASE LINE
VENTRIC. SYSTOLE
AORTIC CLOSURE
A
AORTIC INCOMPETENCY
Upstroke vertical and high. Pointed apex. Abrupt descent. Dicrotic wave almost nil.
B
PULSUS BISFERIENS
Tidal wave marked and high. Dicrotic wave also present.
C
IRREGULARITY
Pulsus alternans. Beats occur in pairs, every second beat small.
D
IRREGULARITY
Bigeminal pulse. Beats in groups of two.
E
IRREGULARITY
Mitral incompetency.
F
IRREGULARITY
Mitral stenosis.
G
UNDULATING BASE LINE
Tuberculous meningitis.

Fig. 71.—Sphygmograms, Diagrammatic and Actual. (Butler.)

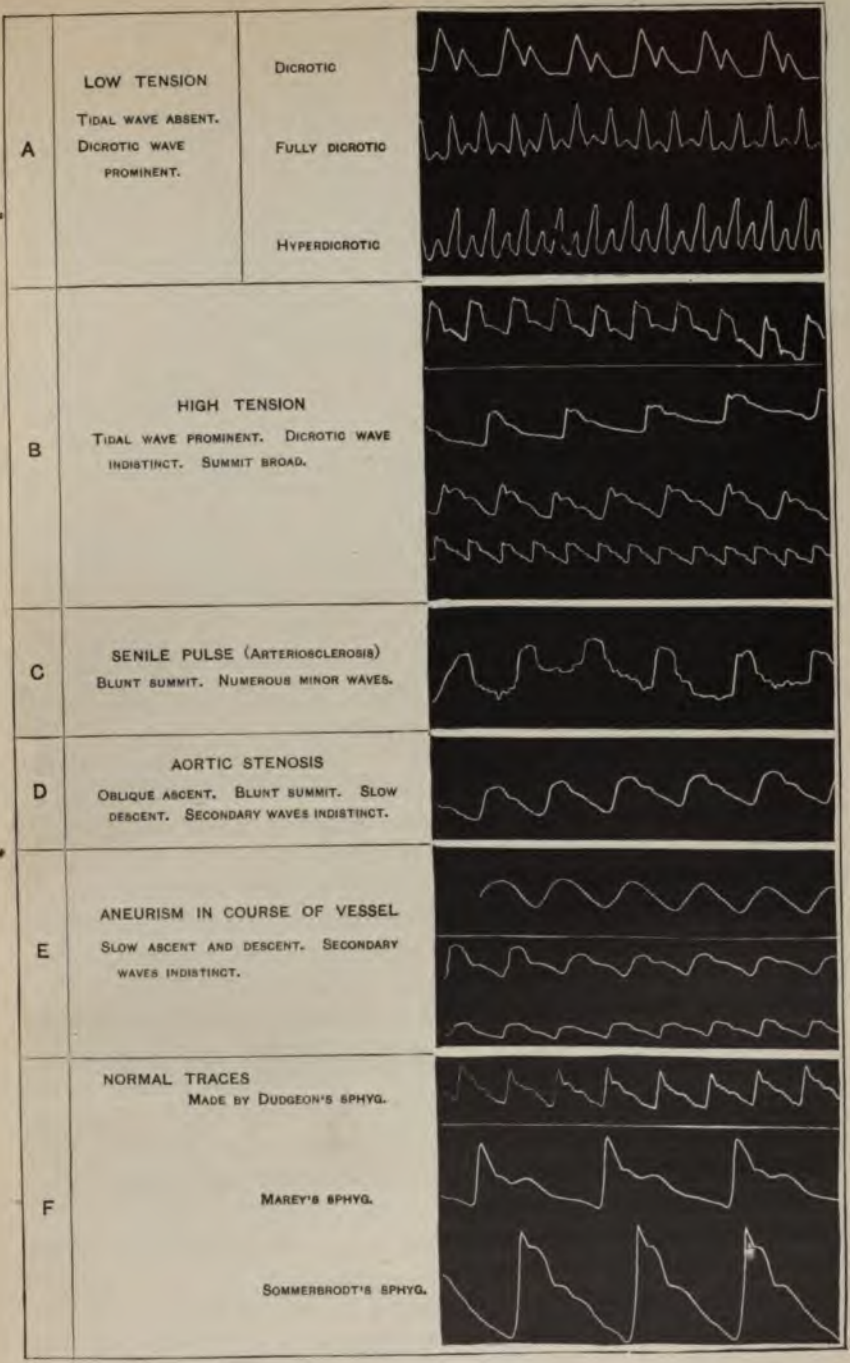

A
LOW TENSION
TIDAL WAVE ABSENT.
DICROTIC WAVE PROMINENT.
DICROTIC
FULLY DICROTIC
HYPERDICROTIC
B
HIGH TENSION
TIDAL WAVE PROMINENT. DICROTIC WAVE INDISTINCT. SUMMIT BROAD.
C
SENILE PULSE (ARTERIOSCLEROSIS)
BLUNT SUMMIT. NUMEROUS MINOR WAVES.
D
AORTIC STENOSIS
OBLIQUE ASCENT. BLUNT SUMMIT. SLOW DESCENT. SECONDARY WAVES INDISTINCT.
E
ANEURISM IN COURSE OF VESSEL
SLOW ASCENT AND DESCENT. SECONDARY WAVES INDISTINCT.
F
NORMAL TRACES
MADE BY DUDGEON'S SPHYG.
MAREY'S SPHYG.
SOMMERBRODT'S SPHYG.

FIG. 72.—SPHYGMOGRAMS, ACTUAL. (Butler.)

of abnormalities of several or all of the quality characteristics described. Not only the frequency of the pulse-beats may be irregular, but their tension, force, size, and duration. Indeed, such inequalities are of graver significance in connection with *weak heart* than mere arhythmia, although general irregularity without serious degeneration of the heart may exist to a degree, in some cases, of mitral stenosis. The alternating and bigeminal pulses, and the *intercidens*, are forms of the *pulsus inequalis*.

(*d*) *Symmetry.*—In the healthy individual, corresponding arteries, such as the two radials, cervicals, femorals, give rise to pulse-waves of like times and symmetric qualities. Hence, in order to determine the presence of local interferences with the arterial circulation, the pulse should be studied comparatively of the different arteries in the same person.

The principal *unilateral abnormality* is a *delay* of the pulse on the affected side; that is, on the side which thus indicates some local obstacle to the current of blood. To palpate both radials, for example, and discover an asymmetry of pulse-impact requires some psychomotor training and skill, which may easily be acquired by practise, however, as in noting the difference in the times elapsing between the carotid and radial, or the carotid and dorsalis pedis pulses in the healthy individual. Besides the delay, there is usually at the same time some *weakening* and *diminution of the size* of the pulsations, as compared with the unaffected artery on the opposite side. In some cases the pulse on one side may be quite obliterated.

In *aneurism* of the aortic arch there may be a marked interval between the time when the pulse-wave reaches the unobstructed and the obstructed radial. Other *causes* of asymmetric pulse are, tumors of the chest cavity, particularly near the axilla; thrombosis and embolism; in pleuritic exudations of large size, and pneumothorax; in surgical conditions of the forearm, and abnormal or anomalous distributions of the arteries.

Thus a delayed, weak, and small pulse in the *right radial* indicates aneurism of the ascending aorta or innominate artery, or of the presence on the right side of any of the preceding disorders mentioned; on the *left side*, aneurism of the descending aorta, especially near the junction with the transverse portion of the arch, and similar lesions, cicatricial contractions, etc., on that side.

Likewise, abnormality of the pulse in one or other femoral or posterior tibial artery may point to thrombus or embolus, or tumor or aneurism affecting the abdominal aorta.

Tortuosity of the Aorta.—As an evidence of atheromatous degeneration, not infrequently a premature peculiarity, one may detect through relaxed abdominal walls, as in women, a more or less serpentine course of the aorta between the epigastrium and the umbilicus. At the same time the aortic walls feel stiffened and resistant to pressure, and the vessel may be pushed from side to side over the spinal column, as if it were a rubber tube. I observed this most recently in a married woman of thirty-four years, who had given birth to one child, has myopic astigmatism, and no evidence of vascular thickening elsewhere. This condition has been well studied in Röntgenography by Sailer and Pfahler.

AUSCULTATION

Normal Arterial Sounds and Murmurs.—The arteries which may be auscultated are, besides the aorta and the pulmonary artery, the carotid, subclavian, brachial, femoral, and rarely the popliteal and posterior tibial, although it is customary to examine the first two only in most instances.

If one applies the binaural stethoscope very lightly over, say, the right carotid artery, with the head slightly extended, one may hear *two normal sounds*, corresponding with the cardiac systole and diastole, and synchronous with the expansion and contraction of the artery. The first sound is weaker and duller, the second stronger and clearer. The former is probably partly the transmitted first cardiac or aortic sound, and partly produced independently in the carotid itself by the sudden stretching of its walls. The second sound is simply the transmitted aortic second sound. Not infrequently the first arterial sound is entirely wanting, especially over more remote arteries, where the transmissions are too weak to be heard. A systolic sound may be audible in many normal individuals along the course of the abdominal aorta, but here the second sound is usually inaudible.

If the stethoscope is pressed upon the artery with sufficient firmness, an *induced* or *pressure murmur* is generated. This may be heard over other large arteries that are so situated anatomically as to be thus compressible. The murmur is systolic, and has a rushing quality of sound which is increased in intensity as the stethoscopic pressure is increased to the point of obliterating the normal systolic arterial sound. There is no diastolic pressure murmur because there is not sufficient backward movement of the blood to create frictional disturbances and vortices.

Abnormal Arterial Sounds and Murmurs.—A harsh systolic murmur replacing the conducted first sound is heard over the carotid in *aortic stenosis.* On the other hand, in *aortic regurgitation* the second sound of the carotid and subclavian is wanting, and the diastolic murmur is faintly heard, if at all. Furthermore, in these arteries not only, but in some cases in the brachial, femoral, and even in the radial and ulnar, and the peroneal and dorsalis pedis arteries, *single systolic sounds* may be audible with the lightest pressure of the stethoscope. Again, with considerable pressure, a *double murmur* (Duroziez's) is heard over the femoral artery in certain cases of aortic insufficiency. The single sounds are probably produced by the sudden and abnormally forcibly tense filling of the arteries by the huge, hypertrophied heart, at a time when they are abnormally relaxed because of centripetal as well as centrifugal emptying.

A *double sound* as well as double murmur may be heard over the crural artery in some cases of *aortic regurgitation,* of *mitral stenosis,* in *lead-poisoning,* and in *pregnancy* (Gerhardt). The second sound is caused by the sudden collapse of the artery, with a large, quick pulse. The second part of the double-pressure murmur of Duroziez is attributed to the short reflux of blood in aortic insufficiency. These embrace the principal *sounds of conduction* in the arteries.

Murmurs Due to Disease of the Arterial Walls.—In the aorta, roughening of the inner coat of the arch, due to chronic *aortitis* in old men, may produce a murmur audible over the carotids and subclavians by transmission. Or it may be caused by narrowing of the lumen by thick calcareous plates, or by pressure from without, as from *mediastinal tumors, enlarged lymph glands,* or an *aneurismal sac.* Localized *endarteritis* of the larger arteries may also be suspected, as where a soft systolic murmur is heard over a portion of the crural artery, for example; or the double murmur of pressure may, when heard over one of the larger arterial trunks, indicate an endarteritis or tortuosity there.

Murmurs Due to Low Tension.—In cases of marked relaxation of the vessels, as from *congenital hypoplasia,* or *fatty degeneration,* or chronic disease, the arteries, especially the innominate and carotids, show increase of caliber, but soft pulse, and on auscultation often reveal soft systolic murmurs.

Anemia and Chlorosis.—These conditions of altered blood composition are usually accompanied with the so-called anemic, hemic, or functional arterial murmurs. They are also systolic in time, seldom loud, although rarely musical, and best heard over the carotid.

The Subclavian Systolic Murmur.—This requires separate and special consideration from what references have already been made to subclavian phenomena. It is a fact that sometimes in health a short, systolic whiffing or blowing sound may be heard over both subclavian arteries, usually stronger on the left side and at the end of inspiration. It is best heard below the clavicle at its outer end. When unilateral, it has a suspicious pathologic significance, namely, of *apical tuberculosis* with *pleuritic adhesions*, the latter pulling or bending the artery, and thus constricting its lumen during the act of inspiration. Even when of bilateral occurrence, the possibility of incipient disease, or of previously formed pleuritic exudation, should be thought of; also, fibroid phthisis with traction, and tumor pressure.

Murmurs in the axillary artery may be due to pressure, especially in women of advanced years, by cancerous enlargement of the lymphatic glands; in early life, by tuberculous glands.

Systolic murmurs may be quite distinct, even loud, over the goiter (thyroid gland) of Basedow's disease; also over the pregnant uterus (uterine souffle). Both of these are of arterial origin.

Finally, may be mentioned the *systolic cerebral blowing* or humming heard really normally over the anterior fontanelle of infants between the third month and the sixth year. This may be audible also over the carotids, and by Jurasz is believed to be the carotid murmur conducted upward, the artery being subjected to compression during the development of the skull.

THE CAPILLARIES

The capillaries give color or complexion, redness or pallor to the skin according to their fulness. The only abnormal capillary phenomenon of diagnostic importance is the *capillary* or *subungual pulse.* This consists of an alternate, rhythmic reddening and paling of the pink zone over the root of the finger nail, corresponding to the quick and extreme filling and emptying of the capillaries, and associated with the large and quick (*celer*) pulse of aortic insufficiency, with hypertrophy of the left ventricle. This alternate waxing and waning of color, synchronous with the cardiac action, is not, however, exclusively significant of aortic regurgitation, but may be visible in several states of decreased blood pressure and low arterial tension. This sign may be elicited similarly by drawing out the lower lip and placing a microscope slide against the mucous membrane so as to produce a slight pallor, which will change to a faint pinkish tint with each systole of the heart. A flush streak on the forehead, caused by sharply drawing the finger across it, may also have corroborative value in the alternate paling simultaneous with cardiac diastole.

THE VEINS

As with the arteries, so with the veins, consideration is unavoidable in connection with cardiac and, to a lesser degree, pulmonary diseases. The phenomena of the venous circulation have diagnostic significance as related to cardiac affections, whether they occur as general venous involvement or in a local way. Thus we find *general venous distention* whenever the return flow of venous blood as a whole is hindered, particularly from failing right ventricle, the result of mitral or tricuspid valve defects, or of relative tricuspid insufficiency because of interference of the pulmonary circulation due to emphysema, chronic asthma, and extensive fibroid phthisis. Here we have associated more or less cyanosis and general edema. *Local venous distentions* and *abnormal pulsations* will be considered as more especially within the scope of this book, as found on the thorax and neck. In fact, the *jugulars* are the most conveniently located and amply sized of all the veins whereby the condition of the right heart may be estimated fairly; indeed, it may be said that the signs of the jugular veins are at once the most available and reliable indices of the venous circulation in general.

INSPECTION

The pathologic phenomena observed on inspection of the veins are: (1) *Engorgement*, and therefore *enlargement*, of the superficial veins; (2) *pulsations* or *undulations*, having respiratory or cardiac causation.

Fulness of the Veins.—In health the prominence of the cervical veins depends partly upon the amount of subcutaneous fat and partly upon the degree of physiologic activity, other things being equal. Of course, the position of the head and the depth and period of the respirations are also influencing factors. Of the two jugulars, only the external, coursing obliquely over the sternocleidomastoid muscle, and the right better than the left, is distinctly visible; the internal jugular, underneath this muscle, and its bulbous junction with the subclavian vein, are not visible in health. In fat-necked individuals, even the external vein may not be seen, notwithstanding the head is placed low and turned toward the opposite side to bring it out, unless the habit or physical temperament is that of the full-veined plethoric. The veins show better in men than in women, and in those who lead active, energetic lives. Also, they are fuller during such acts as coughing, laughing, running, and the like.

Abnormally distended jugulars are evidence of any form of valvular or pulmonary disease causing obstructed circulation through the lungs, and consequently tricuspid regurgitation. Or they may be produced by aneurismal or neoplastic (mediastinal) compression of the superior vena cava or innominate vein. In the case of mediastinal tumor there may be suggestion of it in the presence over the sternum of a few single engorged veins of small size. In marked cases of engorgement the jugular bulb is also seen swelling out ¼ to ½ in. above the sternoclavicular articulation from behind, and between the divided insertions of the sternocleidomastoid muscle.

A point in the diagnosis of cervical vein engorgement, as differentiated from a merely physiologic or plethoric fulness, may be aided by noting the susceptibility of the former abnormal condition to respiratory movement. The pathologic turgidity is usually distinctly lessened during inspiration and increased during expiration, while normally this difference of undulatory rise and fall is hardly perceptible.

Venous Undulations and Pulsations. (1) THOSE CAUSED BY RESPIRATORY MOVEMENTS.—*Normally*, the continuous flow of blood in the veins, as manifested in those of the neck while collateral attention is also given to the movements of forced respiration, shows an unmistakable filling during expiration, and a relative collapse during inspiration, because of the negative intrathoracic pressure at that time which draws the blood toward the heart, the swelling of the jugulars during expiration being due to the positive pressure exerted upon the veins then, which causes "a retrograde wave of blood to close the valve above the jugular bulb."

Pathologically, an aggravation of the jugular undulations accompanying the movements of respiration are significant of *chronic asthma* and *emphysema*; particularly in the latter disease, where the swelling of the cervical veins, while the patient is lying down, is a conspicuous feature. Coughing increases the turgidity of the undulations markedly. In the rare affection of *fibrous mediastinopericarditis*, a reversal of this phenomenon takes place; that is, the jugular veins are filling during inspiration, because of the bending of the superior vena cava by adhesions during that act, thus hindering the venous flow in the direction of the heart.

(2) THOSE CAUSED BY CARDIAC CONDITIONS.—*In health,* the *venous pulse* is a gentle undulation, best seen in the external jugular vein in thin persons. The wave rises and passes just before the cardiac systole, and hence (*a*) the *normal* venous pulse is *presystolic* or *negative.* (*b*) The *pathologic* modification of this pulse makes it

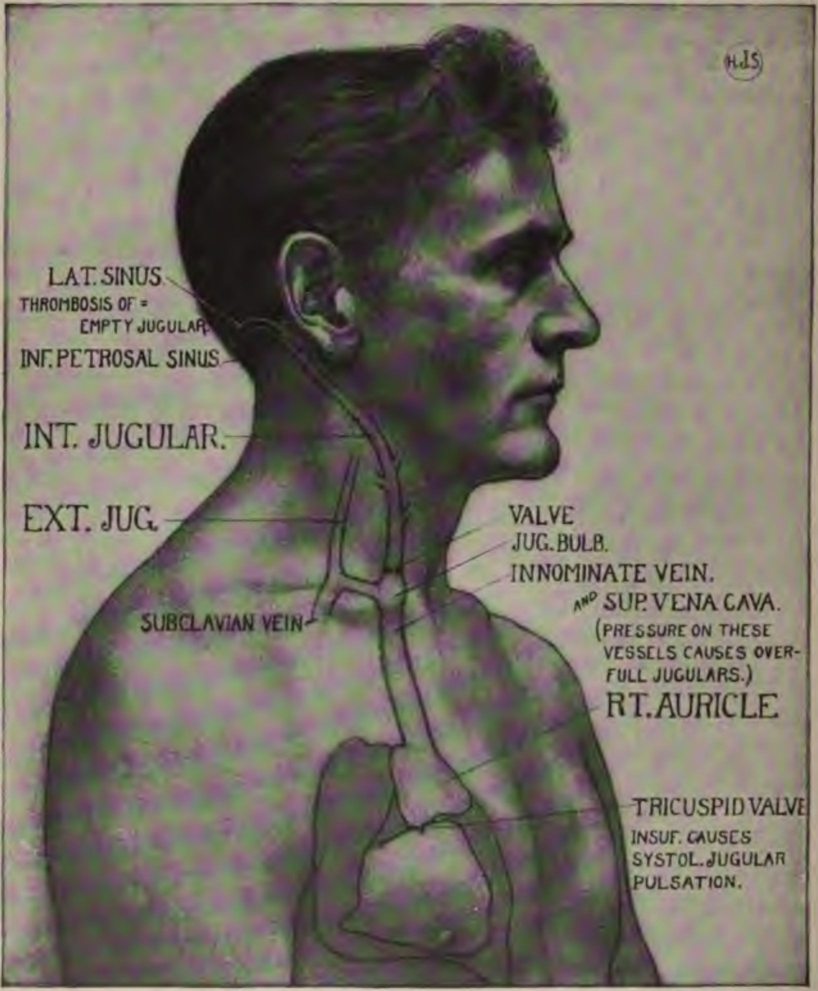


FIG. 73.—DIAGRAM OF THE RIGHT EXTERNAL AND INTERNAL JUGULARS, SHOWING THE MECHANISM OF JUGULAR COLLAPSE, DISTENTION, AND PULSATION. (Butler.)

systolic in time, or *positive.* Obviously, it is important not to confuse the systolic venous with the synchronous carotid pulse. A pulsating jugular may be due to the transmission of the carotid impulse. If so, then "milking" the vein upward will not be followed by a venous wave from below, as is the case with a positive venous pulse of *tricuspid regurgitation,* which is almost always the cause of it.

(*a*) The *normal or negative venous pulse,* as seen in the external jugular, should not, as one author writes, be designated as a kind of undulatory collapse of the vein occurring with the systole of the heart, although that does actually happen. For the complete filling of the vein, which makes it rhythmically visible before its emptying, is presystolic, and hence negative, since it is not due to a positive or systolic wave of blood *from the heart.* This may be demonstrated by compressing the vein near its middle with the finger, whereupon pulsation ceases on the proximal side, showing that the blood does not regurgitate from the heart; and at the same time there is a decided diminution of undulation on the distal side, showing that the pulsation is not transmitted from the carotid. In observing the normal jugular pulse, it is characteristic to see the presystolic wave rise slowly, to be followed by a sudden systolic collapse, then a short but appreciable interval before the next wave. The phenomenon is due to the systole of the right auricle; that is, since the auricle contracts during venous distention—both at presystole—the back current caused is stopped at the jugular valve, which transmits the shock above. The negative venous pulse may also be distinguished from carotid pulsation not only by the time, but by the sensation to the finger of undulation rather than circumscribed impact, by the "impression of passive force rather than of active power," and by the greater anatomic extent of wave.

The presystolic venous pulse is often prominently visible in chlorosis.

(*b*) *The positive or pathologic pulse,* synchronous with the apex-beat and carotid impulse, is best seen at the right jugular bulb, in the sternocleidomastoid fossa. Should the valve here in the internal jugular become incompetent, the systolic, positive venous pulsation will then be visible upward in the neck. This sign is pathognomonic of *tricuspid regurgitation,* the reflux blood current being urged upward through the incompetent orifice into the auricle with each contraction of the right ventricle. Thence it passes into the superior cava, right innominate, and internal jugular veins to the bulbar valve, which soon ceases to stand the strain, and itself becomes incompetent.

The jugular pulse may be absent in the upright posture, as gravity favors its appearance.

Systolic venous pulse may appear later on the left side in severe cases of tricuspid regurgitation; and, on the other hand, when marked enfeeblement of the heart, or extreme rapidity, ensue, the pulsations may quite disappear.

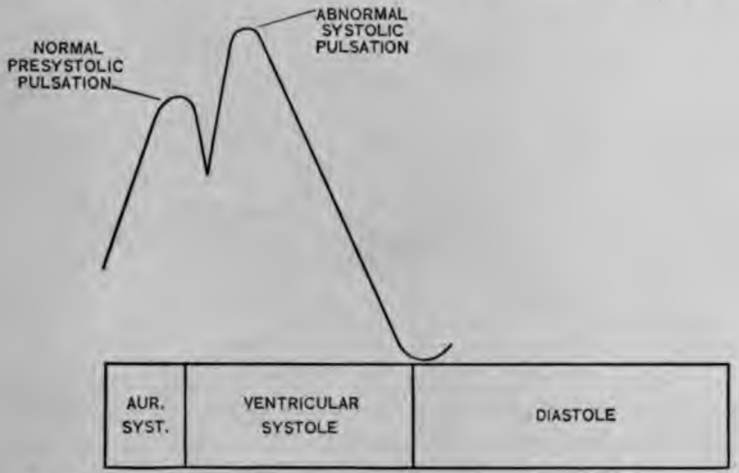


Fig. 74.—Diagram showing the Presystolic and Systolic Jugular Pulse. (Butler.)

A positive centrifugal venous pulse may exist temporarily as the result of functional, muscular, tricuspid insufficiency connected with pronounced anemia. This often appears at the same time as the soft systolic murmur of a relative mitral insufficiency, thus differing from the tardily appearing tricuspid insufficiency secondarily due to an organic (endocarditic) mitral defect (von Leube).

PALPATION

Palpation may have to be brought to the aid of inspection in some cases where by the latter method alone the diagnosis of positive venous pulsation may be difficult, doubtful, or impossible. Thus, it must be decided first that the pulsation is really systolic, and then that it is intravenous in origin, and not transmitted from the adjacent carotid. The former may be determined, of course, by palpating the apex-beat and noting its synchronism with the jugular pulse. If, owing to rapidity of cardiac action, there is doubt as to the systolic time of a venous pulsation, the finger may be placed upon the vein at the root of the neck, and slid upward with sufficient pressure to empty it. Immediate filling and pulsating would indicate incompetence of the jugular and tricuspid valves, whereas competency of these valves would be accompanied with a distinctly slower refilling of the vein from its small collaterals. The phenomenon is also less likely to be altered by deep respiratory action than is the normal or negative pulse.

Transmitted carotid impulse may also be differentiated from true venous pulsation of systolic time by stroking or "milking" the vein from below, or by placing the finger midway between the clavicle and ear, which, if the pulsation is *imparted* or *false*, will cause a distention of the vein and exaggeration of pulse above, and a corresponding emptiness and collapse below. But if the pulsation is actually in the vein, from a tricuspid insufficiency, the filling will be noticed as before.

In some cases, instead of a single venous throb, synchronous with the systole of the heart, there may be a double wave, the presystolic being weaker than the systolic wave.

The systolic venous pulse may be simulated, rarely, by a sudden *diastolic collapse* of the jugulars due to the sudden diastole of the heart aspirating the blood from the veins, after a labored systole caused by extensive pericardial adhesions.

Palpation may be of further service in aiding inspection of the veins, as in determining whether a pulsation is carried beyond the sinus of the jugular vein. Thus, in tricuspid regurgitation, a systolic thrill may be felt over the position of the incompetent valves. Again, in rare cases, the backward undulation may be visible and palpable in the thyroid, external jugular, and facial veins, or a pulsation may be propelled into the larger venous trunks of the arms, as the axillary, brachial, and cephalic and basilic veins. Moreover, it is not very unusual to find, in such instances, a venous pulsation propagated down the inferior vena cava into the veins of the liver, so that palpation of that organ detects a rhythmic pulsation—the *venous liver pulse* —following closely the apical stroke. The systolic swelling and diastolic collapse may be the more evident because of the congested condition of the organ which is common in these cases, with moderate enlargement. This sign is best elicited by grasping the whole right lower zone of the chest, one hand being placed with the fingers below the tenth, eleventh, and twelfth ribs behind, and the other in front over the hypochondrium and epigastrium.

In the *diagnosis* of the systolic or positive venous pulse, it should be pointed out here that besides tricuspid insufficiency, it may be indicative of combined mitral insufficiency and patulous foramen ovale, or of the extremely rare varicose aneurism of the thoracic aorta communicating with the superior cava.

Venous Pulse of Quincke.—This has been described as observed particularly in the superficial veins of the hand and the back of the foot, in connection with aortic insufficiency and the capillary pulse. It represents probably the arterial pulse propagated through the capillaries; hence the term, also, of *progressive venous pulse.* This pulsation may be associated with simple relaxation of the arteries, with phthisis, neurasthenia, and anemia, and is reported to have been seen in health (?).

A *venous thrombosis* may be detected on palpation in one of the larger veins of the extremities. The veins feel firm and cordlike, and are usually surrounded by some local edema. This is not a rare complication of typhoid fever, septic infections, and of senility.

AUSCULTATION

The only phenomenon of importance is the venous hum or *bruit de diable* heard over the jugulars. This murmur may be physiologic, stethoscopic, or pathologic in origin; usually the last. The jugular bulb sound of Bamberger will be referred to shortly.

The Venous Hum.—This is a soft, continuous humming or blowing sound heard over the jugular veins, but with greater distinctness on the right side. At times the sound has a buzzing or even musical quality. As it occurs almost exclusively in the internal jugular vein, it is best heard in the hollow above the clavicle and between the origins of the sternocleidomastoid muscle, right over the bulbus. That the murmur is venous and not arterial can be demonstrated by pressing firmly on the dilated lower end of the jugular vein, just above the clavicle, when the hum will disappear at once. Again, pressure upon the carotid higher up on the neck does not alter the character or intensity of the venous murmur.

The occurrence of the venous hum *physiologically* is accounted for mostly by the anatomic conditions. In the first place, it may be due to the rush of blood from the narrow vein into the wider chamber of the jugular bulb; or, and in addition, as Hamernjk has shown, it may be attributed to the considerable bend which the internal jugular vein makes in a forward direction, in front of the transverse process of the sixth cervical vertebra, and to the pressure of the traversing omohyoid muscle. Doubtless the vibrations are at least partly due to the venous walls themselves. By turning the patient's head to the opposite side, and thus putting the muscles on the stretch on the side under examination, the slightly audible hum may be increased in loudness.

The *intensity* is influenced by other conditions. The pressure of the stethoscope develops or augments it. Likewise, anything which accelerates the flow of blood from the jugulars, as during deep inspiration, the standing instead of the sitting or lying posture, the period of the heart cycle—since, naturally, the murmur would be weaker during the cardiac systole, diastolic intensification may be noted. To detect the venous hum, or variations in its intensity, it is well to have the patient hold his breath, so as to avoid the confusing tracheal sounds. The murmur is sometimes heard over the upper part of the sternum, and may even be traced as far as the aortic area in certain instances.

Pathologically, the venous hum is commonly significant of the anemias, especially of chlorosis and pernicious anemia. In some rare cases a venous murmur may be heard over the *femoral veins*, and this may be intensified by elevating the limb. In weakened and varicose conditions of the femoral veins, or when the valves are insufficient or absent, a sharp, whizzing murmur may be excited by violent coughing, and may even be palpable over the femoral vein, just below Poupart's ligament. It is probable that the venous hum of anemia is caused largely by the vibrations set up on account of the diminished quantity and plasticity of the blood, the currents being thus more readily and more forcibly produced.

Intermittent venous hum has been distinguished from the continuous by Weil, Guttmann, and others. It is of comparatively rare occurrence, however. It may be indicative of augmentation of the jugular velocity of blood due to inspiration, to cardiac diastole, or to both combined.

Jugular sound, heard over the bulbus, as discovered by von Bamberger, signifies the shock against the valve in consequence of the reflux wave of a tricuspid insufficiency. This venous sound passes into a murmur when the venous valve becomes incompetent. Rarely such valve sounds may also be heard over the femoral veins, in the exceptional instances when the recurrent waves reach these vessels. As pointed out by Friedreich, the sounds may be double, presumably due to auricular as well as ventricular contraction.

SECTION IV

SPECIAL PHYSICAL DIAGNOSIS

CHAPTER XV

DISEASES OF THE HEART, INDUCTIVELY CONSIDERED

Actual and hypothetical cases of heart disease will be narrated in brief, typical, and suggestive symptomatic outline. The physical signs will then be stated, and the method of analysis, and direct and differential diagnosis followed as in practical procedure.

Case No. 1.—A day-laborer, aged forty-six, was admitted into the hospital ward. He walked, complaining of and manifesting shortness of breath, some precordial distress, though not actual pain, and palpitation of the heart. The dyspnea was quite marked on walking rapidly or going up-stairs, and for two weeks his work as a dirt-shoveler bore so hardly upon him that he had to quit it. He showed anxiety and nervousness of countenance and manner, and weakness of attitude and musculature. His lips and cheek eminences had a faint bluish tint, and his nostrils dilated visibly with labored inspiration. He gave a history of having had acute inflammatory (articular) rheumatism for six weeks, when nineteen years old, and a mild attack for two weeks just four months previous to admission. His health and strength had been generally good until the last attack, but since then he had noticed a slight, progressive loss of health and endurance, frequent discomforts after eating, with occasional eructations and nausea, constipation, a little cough after exertion, and momentary dizziness when fatigued. He was a "moderate" user of beer, whisky, and pipe-tobacco.

Physical Signs.—*Inspection* of the precordium showed no undue prominence or depression. There was, however, a diffuse, wavy impulse in the third, fourth, and fifth interspaces, to the left of the sternum, and distinct pulsation in the epigastrium. The apical impulse was also somewhat increased in area, and displaced to the left, the center being about ½ in. beyond the nipple (midclavicular) line, in the fifth interspace.

Palpation.—This confirmed the information obtained visibly, and added the following signs: The precordial and apical and epigastric pulsations were also feeble; the apex could be located more positively by the finger-tip near the middle of a moderately diffuse and weak impulse; slight impulse was noted also in the second left interspace near the sternum. There was a perceptible but faint systolic thrill at the apex. The pulse was diminished in volume, moderately weak or small, the tension lessened, and a trifle irregular as to time, volume, and force.

Percussion.—The cardiac area of dulness (deep or relative) was increased in all directions: to the left, ½ in. beyond the nipple-line in the fifth interspace, and to the nipple-line in the third and fourth interspaces; to the right, a trifle beyond the right border of the sternum; above, it begins with absolute dulness in the third interspace, parasternal line, thus making a broad and rounded upper and outer border of dulness; below, there could be demonstrated a diminution of sternal resonance at the base of the ensiform process.

Auscultation.—Both heart sounds were weakened, the first relatively more than the second, which could be heard quite distinctly at the apex. The pulmonic second sound was clearly accentuated in intensity, and of equal pitch with the aortic sound. The first sound was partially obscured by a soft systolic murmur, maximum intensity over the apex; the loudness was increased when the patient assumed the dorsal position. The murmur was audible nearly as far as the left sternal border, quite so up to the third interspace, and was transmitted outward as far as the anterior axillary region, with rapidly diminishing intensity. Once or twice, after some exertion in rising quickly, the murmur was heard faintly near the angle of the left scapula.

DIAGNOSIS FROM THE ANALYSIS OF THE PRECEDING.—In the first place, that we are dealing with a heart case is inferred from the *rheumatic history* and the gradual onset of *breathlessness,* aggravated by exertion, along with palpitation, precordial distress, and slight cyanosis. A history of articular rheumatism in itself should point to the probability of a chronic valvular endocarditis, from its well-known frequency as the principal cause. The novice might be inclined to surmise the case to be one of *spasmodic* (*bronchial*) *asthma;* but the sudden, brief, sharp, *expiratory* character of the latter form of dyspnea, its frequent occurrence at night, the prominence of cough, its intermissions, the absence of local cardiac symptoms and of a cardiopathic history, serve to distinguish this from the so-called cardiac "asthma."

Secondly, we infer moderate enlargement of the heart because of the displaced apex-beat and increase of the area of dulness.

Thirdly, this enlargement is most probably caused by dilation rather than hypertrophy, at least by the predominance of dilation, because of the diffuse impulse and outward rather than downward displacement of the apex; since such broad impulse, laborious in manner, with weak, small pulse, is evidence of ventricular weakness associated with consequent yielding of the walls. In hypertrophy, too, the apex would be more downward, and concentrated in area of impulse.

Fourthly, the enlargement or dilation involves the two ventricles and the left auricle from the facts of percussion, inspection, and palpation, namely, outward and inward extension of dulness, as well as upward to the left of the sternum; the apical impulse displaced to the left (left ventricle); diffuse impulse below the third rib, into the epigastrium (right ventricle), and impulse in the third left interspace near the sternum, which may, however, be attributed partly to a dilated, throbbing pulmonary artery, as well as left auricle.

Fifthly, by auscultation alone we determine an *insufficiency of the mitral valve* from the facts that the murmur is best heard at the apex, where mitral murmurs have their maximum intensity; that its systolic rhythm occurs at the time when the mitral valve should normally be closed, and that its propagation into the left axilla means a regurgitation of blood backward from the left ventricle into the left auricle.

A rational interpretation of the *physical pathology* from the physical signs confirms the diagnosis of a mitral leakage (chronic), and thus explains the anatomic and functional effects of the lesion. Thus:

(1) The left auricle, the chamber which receives the regurgitating blood, is naturally subjected to increased pressure, receiving also, as it does, the normal flow from the pulmonary vein. This produces first a dilation, then compensatory hypertrophy, then more dilation, to be succeeded perhaps by more hypertrophy and dilation, and so on until permanent enfeeblement and dilation of the auricular walls ensues; hence the extension of cardiac dulness over that region.

(2) While hypertrophy of the left auricle predominates, so that the increased quantity of blood received during diastole is forcibly

returned to the left ventricle by the auricular systole, the effect may be felt somewhat by the ventricular walls, which then respond to the extra labor by beginning hypertrophy also. The physical evidence of this, which is limited practically to a slight outward displacement of the apical impulse, is seen only in early cases.

(3) Next, as auricular weakness and dilation become extreme, backward pressure into the pulmonary veins occurs; engorgement of the pulmonary capillaries and arteries follows, and the result is dilation from overdistention of the right ventricle, and more or less prompt and effective hypertrophy, which after a long interval passes into marked weakening and dilation of its walls in turn. Therefore the diffuse, flabby impulse over the lung-exposed portion of the heart, the marked epigastric pulsation, and the cardiac dulness to the right of the sternum.

(4) During the predominance of right ventricular hypertrophy, however, the blood is forced forward with proper flow, so that, again, an overdistended left auricle is throwing augmented strain upon the left ventricle, which then progressively enlarges in hypertrophic dilation, and thus accounts for left (and often slightly downward) apical displacement, and increased area of cardiac dulness to the left.

(5) In the last stages (which the patient's case here cited does not present), the dilation of the right ventricle to a sufficient degree causes leakage of the tricuspid orifice (relative insufficiency); the flow of blood from the right auricle to the right ventricle is impeded; dilation, slight hypertrophy, and again dilation of the auricle follow, and the visceral and peripheral venous congestions, as of the liver and portal circulation, legs, etc., become gradually manifest, with consequent dropsical effusions into the peritoneal sac (*ascites*), and the edematous swellings so characteristic below the knees, with smooth, shining, bluish pallor of skin, and the pit marks of finger pressure.

DIFFERENTIAL DIAGNOSIS.—Mitral regurgitation is seldom confounded with other valvular lesions, although the murmur of aortic obstruction or roughening, or of tricuspid regurgitation, may simulate that of the mitral lesion. But the associated physical signs, and the bearing of the etiologic history and secondary effects of the valvular derangement serve at once as a check upon or confirmation of the indication of the murmur alone. The diagnosis cannot properly or precisely be made by the detection of a murmur merely.

Functional murmurs, and those of *relative mitral insufficiency*, are more likely to simulate the organic mitral defect than the organic affections of the other valves. The principal points of differentiation have already been described in a general way. It should be remembered, however, that the *functional* murmurs are usually much softer and more variable in loudness than the organic; that, although they may be audible inside or outside the apex-beat (Potain), more often they have their maximum intensity at the pulmonic area; that they are not associated with enlarged area of cardiac dulness or secondary pulmonary or general venous congestions or accentuation of the pulmonic second sound, and that they are localized instead of transmitted. The murmur of *relative mitral insufficiency*, which sometimes occurs in the course of acute febrile diseases from temporary weakness of the myocardium, may be mistaken for the endocarditic lesion at the same orifice. During the course of acute inflammatory attacks, in particular, it is impossible to determine whether a systolic murmur is due to relative insufficiency at the auriculoventricular orifices or to the beginning of a structural valvular change causing leakage. Even later, for some time during and after convalescence, anemia may cause a soft, blowing murmur simulating an incipient organic mitral regurgitation, until the cure of the blood deficiency causes the former to disappear and clear the diagnosis.

Other conditions in which a relative mitral regurgitant murmur may occur should be mentioned in connection with the resources of differential diagnosis. Such are fatty heart, cardiac enlargement of chronic Bright's disease, myocarditis, and toxemic disorders. These murmurs are also variable in intensity, being especially affected by respiration and posture, and often disappearing, temporarily at least, under treatment with digitalis. Also the heart sounds are more frequently and decidedly weak and irregular. A relative insufficiency murmur at the mitral orifice may, it is true, be superadded to a real organic lesion of the same, but the latter is sure to remain after the therapeutic test just referred to.

From *aortic stenosis*, which also has a systolic murmur, mitral regurgitation may be differentiated by the fact that the murmur produced by the former lesion, even when heard pretty much all over the precordium, nevertheless has its area of greatest intensity at the second right interspace; that it is not likely to be transmitted to the left axilla, still less to the left scapula, but rather is well heard up in the carotids, where the mitral murmur is inaudible; that it is usually harsher in quality than the latter; and that only in the very later stages is it accompanied with secondary venous congestive effects in the pulmonary and peripheral circulations (from relative mitral insufficiency due to the prolonged strain upon the left ventricle, and consequent dilation).

Tricuspid regurgitation produces a systolic murmur, heard also at the apex, but louder near the sternum and an interspace higher, while the mitral murmur diminishes in intensity in that direction, and is heard with distinctness beyond the left border of the heart. If any error is made it is rather in failing to detect the concomitant tricuspid murmur, which is practically always secondary to mitral disease. When present, however, there is less likely to be accentuation of the pulmonic second sound; and usually associated, on the other hand, the venous phenomena already described in the preceding chapter.

Having thus differentiated, and excluded the possibility of confusing, pericardial and cardiorespiratory murmurs (*q. v.*), the *diagnosis* of organic *mitral insufficiency* is made.

In examinations for life-insurance it frequently happens that a very slight degree of mitral regurgitation is discovered on auscultation, but which, so far as symptoms are concerned, the applicant is unaware of, and which may not be signally aggravated to loss of myocardial compensation for fifteen or twenty years in a person of early or middle life, and of moderate habits and work and tranquil mentality.

Case No. 2.—A slender, almost phthisical-looking woman of twenty-seven years, with a history of having had several rheumatic attacks since childhood, complains of rapidly increasing breathlessness and palpitation of the heart within a month of consultation. These symptoms were especially distressing on exertion, and were accompanied with pain in the left side of the chest, and a short, dry cough. The face is pale, with a bluish duskiness of the cheeks, lips, and slightly clubbed finger-tips. The insteps and ankles are slightly edematous. Dyspepsia (sour eructations being troublesome), constipation, scanty urine and menses, vertigo, and sleeplessness are also complained of. A bronchitis was suffered from the previous winter, and twice blood-tinged expectoration was noticed.

PHYSICAL SIGNS.—*Inspection* of the precordium notes a decided bulging of the lower part of the sternum, and of the fourth interspace, fifth rib, and fifth interspace, over the normal position of the right ventricle. In the same region there is visible pulsation, diffuse and fluttery; also, slightly, in the second and third left interspaces, but a little more prominently in the epigastrium. The *apex impulse* is just noticeable, and not displaced.

Palpation.—By this method the visible signs are confirmed: over the body of the heart, where the diffuse pulsations are noted, the hand feels a wavy fluttering, with an occasional laborious heave; the epigastric pulsation is palpable, and a slight impulse is felt in the second left interspace near the sternum. In addition, the apex-beat is perceptibly weak, and felt in the fifth interspace in the midclavicular line. Characteristic, however, is a thrill, presystolic, felt over the apex; it has a short, slightly rough, purring quality, running up to and terminating abruptly with the apex-beat, which follows immediately as a sharp shock or thump. The thrill is more marked during expiration.

The pulse is distinctly small, soft, weak, and irregular in regard to the rhythm, thus being synchronous with the cardiac impulse, which in its irregularity seems at times to be a prolongation of the thrill.

Percussion.—The area of deep-seated cardiac dulness is increased transversely, especially to the right, as far as one inch beyond the right sternal line in the third, fourth, and fifth interspaces; upward along the left edge of the sternum, a few fingerbreadths beyond in extent, as far as the second interspace. Dulness is also marked over the lower half of the sternum, and in the epigastrium around the xiphoid.

Auscultation.—The heart sounds are weak at the base, and somewhat irregular in rhythm. The first sound at the apex is short, valvular, slapping in character, and preceded by a rough, blubbering murmur. The second sound is very distinct at the base, and especially loud and accentuated at the pulmonic orifice. On the other hand, the aortic sound is actually and relatively weak. Both sounds are heard separately on account of a barely perceptible interval; that is, the second is reduplicated or divided. The diastolic interval between the second and first sounds seems to be prolonged.

The murmur is a long one, beginning early during diastole, but reaching its climax of roughness and intensity just before ventricular systole—presystolic, therefore, and synchronous with the thrill. It is practically limited in audibility to an area about the apex not more than two and one-half inches in diameter, and is not transmitted. The murmur sounds very like a whispered roll of the letters *r-r-r-b*, stopping suddenly with the sharp, tapping first sound.

ANALYSIS.—*First*, we have here evidently a serious *organic* cardiac affection, judging from the rheumatic history in conjunction with the symptoms, subjective and objective, of impaired and unbalanced circulation.

Secondly, there is undoubted dilation of at least two of the cardiac chambers, namely, the left auricle and the right ventricle, because of the location and character of the pulsations noted by inspection and palpation, and because of the percutory outlines of dulness. At the same time, important negative evidence as to the left ventricle is to be considered in respect to the absence of dulness to the left, and the position and weakness of the apex-beat.

Thirdly, the presystolic thrill and murmur localized at the apex indicate the valvular or orificial difficulty to be mitral, and obstructive in nature, because these signs occur at the time when, normally, this auriculoventricular opening is free for the blood to pass from the left auricle into its fellow ventricle, but in this case is not so, by reason of the palpable and audible disturbances set up at this time.

The DIAGNOSIS, therefore, is *mitral obstruction* or *stenosis.*

The PHYSICAL PATHOLOGY is similar to that of the preceding case of mitral regurgitation, so far as the dilation of the auricle and ventricle are concerned. Here the pulmonary circulation is congested because the blood is prevented from getting into the systemic circulation except under great difficulty, being held back from the left ventricle by the mitral constriction, whereas in the other instance it was forced backward through the leaking valve by the contracting ventricle.

Hence, too, since the left ventricle in mitral stenosis has less than the normal quantity of blood to circulate, it is inclined to atrophy and shrink behind the right ventricle, which is hypertrophied from too much work to do. That the pressure in the pulmonary circulation is also above normal is indicated by the accentuation of the pulmonic sound, and especially by the comparatively higher pitch than the aortic, showing the relatively lower tension in the aorta. That the left ventricle is incompletely supplied with blood is likewise evidenced by the weak apex-beat and the small and weak pulse.

The fact that the full intensity of the murmur is heard just before systole, although somewhat audible at the middle of diastole, proves obstructive difficulty at the mitral orifice, since at this latter portion of diastole the auricular systole produces vibrations of sufficient strength to be well heard, while the moderate speed with which the blood flows through the narrowed orifice in the first half of diastole is insufficient to cause such vibrations.

DIFFERENTIAL DIAGNOSIS.—*Mitral regurgitation* may be associated with mitral stenosis, but the murmur may be recognized by its being softer and lower in pitch, beginning with the first sound and diminishing, as well as being transmitted to the axilla, instead of having a crescendo of intensity up to the first sound, and then ceasing abruptly. Presystolic thrill is more common than the systolic thrill of insufficiency also. In mitral stenosis, doubling of the second sound is not only more common than in mitral regurgitation, at the base, but is not infrequently, as an apparent doubling, heard over the mitral area as well; the causation of the latter, however, is different from that of the former. Again, in mitral obstructive disease there is more extension of dulness to the right than in the regurgitant form, and none to the left as in that. Finally, the pulse is decidedly more diminished in volume and force in stenosis, and arhythmia is more marked.

Tricuspid stenosis produces a murmur heard almost in the same situation as that of the mitral lesion, and having an identical time and quality. But the rarity of the tricuspid affection seldom calls for its differentiation.

Aortic regurgitation has a diastolic murmur beginning with the second sound, which the mitral stenotic seldom does, and dies away, while the latter increases up to the first sound. Again, the aortic regurgitant murmur is usually very soft and smooth in quality, heard near the second right interspace, and transmitted down along the sternum, although cases sometimes occur where a typical presystolic murmur of rolling quality, with thrill, is nevertheless shown post mortem to have been caused by aortic regurgitation, and not mitral stenosis—the so-called "Austin Flint murmur." Here the diagnosis may be made by noting whether there is marked enlargement of the left ventricle, which would point to aortic insufficiency, especially if at the same time there is weak second sound and loud first sound, the reverse of mitral obstruction, which has a short, snapping first and an accentuated and often reduplicated second sound. The differences in the areas and directions of extent of cardiac dulness, and of the positions of the apex-beat, are also important.

Pericardial friction, especially when due to adherent pericardium, may simulate mitral stenosis; but the murmur is seldom limited to presystolic time, has a superficial, short, scratching sound, and is inconstant; the first sound at the apex, also, is weakened or indistinct instead of accentuated and flapping in character.

Case No. 3.—A large, heavily built Irishman, teamster, fifty-one years of age, came into the office much oppressed with dyspnea, which he said came on rather suddenly about a month or six weeks previously. He was flushed with and smelled of whisky, which he admitted he had been in the habit of using daily for thirty years, and said that at other times persons told him he was pale. He had always worked hard at heavy lifting and the like, and remembered to have felt the effects of physical strain recently in a sort of momentary blinding dizziness, sense of suffocation, headache, precordial distress with some neuralgic pains radiating to the left shoulder, followed by faintness, restlessness, sweating, and considerable muscular weakness and general prostration lasting for a day or two. He said that when a young man he had one attack of inflammatory rheumatism, which affected only the left knee-joint, and which he recovered from in about two weeks.

PHYSICAL SIGNS.—*Inspection* shows marked fulness of the fourth and fifth ribs and interspaces and the sixth rib, from the left parasternal line to a little beyond the nipple-line. The apex-beat is moderately diffuse, but with heaving impulse central in the sixth interspace, fully an inch and a half outside the mammillary line, and quite near the anterior axillary. There is visible over the lower half of the precordium, also, a general, slightly labored impulse. Violent pulsations, with exaggerated diastolic collapse of the temporal, carotid, axillary, brachial, radial, ulnar, femoral, and dorsalis pedis arteries, are plainly seen. The larger arteries mentioned are distended and somewhat tortuous, the throbbing temporals being very serpentine.

Palpation.—The whole hand applied recognizes a strong, widespread cardiac impulse. The apex-beat occasionally conveys the impression of a double muscular movement. At times a faint diastolic tremor is felt at the base of the heart, near the sternum.

The pulse (radial and elsewhere) is peculiarly quick and sharp in wave contact, yet soft, and it is hardly felt before it drops from the finger in as sudden and volatile a manner. The latter phenomenon becomes more distinct when the arm is raised. These facts draw attention to the capillaries, which are examined by inspection, and a capillary pulse is detected beneath the nails. There is an occasional abortive radial pulse-wave.

Percussion.—The area of cardiac dulness extends downward and outward from the third interspace near the sternum, as far as the sixth interspace, and about one and one-half inch to the left of the nipple-line. The dulness extends inward from thence to the left sternal line, from the xiphoid to the third rib cartilage, and all over this enlarged area is marked, showing that there is less than normal amount of lung overlapping, provided there is no consolidation of its tissue.

Auscultation.—The *heart sounds*: the first sound is loud, and yet, paradoxically, peculiarly muffled or lacking in tone; the second sound is very feeble and fugacious at the aortic interspace; the pulmonic sound is distinct, but not accentuated.

Applying the stethoscope carefully, a very soft, blowing murmur is heard at the base of the heart, with a maximum intensity in the third left interspace at the sternal edge, although audible also in the corresponding region of the right side, and at midsternum of the same level. It is synchronous with the second sound, and occupies at least one-half of the diastolic interval following it. The murmur is transmitted down the sternum as far as the xiphoid junction, and to the left as far as the parasternal line in the fourth and fifth interspaces, where it is fairly audible.

Over the large throbbing arteries—the carotids, subclavians, and femorals—a short, snapping sound, systolic; on pressure with the stethoscope this is replaced by a distinct murmur; occasionally, while varying the pressure, a diastolic murmur is also heard.

ANALYSIS.—In the first place, that we are dealing with a heart that is greatly enlarged and most laboriously beating because of severe strain is evident from the position and character of the apex-beat, the extent of cardiac dulness, and the visible pulsations of the principal arteries. For the same reason, we know that the bulk of enlargement is a hypertrophic dilation of the left ventricle. This must mean that there is obstruction or insufficiency at the aortic orifice because of the position of greatest intensity of the murmur discovered. And because that murmur is diastolic, and is transmitted down the sternum, the lesion means a leakage at the aortic cusps, since at that period of the cardiac cycle the valve should be closed. The sound and double murmurs in the arteries, and the quick, short, water-hammer pulse afford additional proof that there is exaggerated ventricular contraction, and sudden diminution of intravascular pressure instead of the gradual, sustained wave which indicates normally that when the aorta is dilated with each fresh column of blood, the valve is able to hold it until the next systole.

The DIAGNOSIS OF AORTIC REGURGITATION is thus made.

The PHYSICAL PATHOLOGY is patent. The constant, rhythmic return into the left ventricle from which they have just come, of regurgitating currents of blood, cannot but issue, as it does, in enormous strain, since this chamber is also as regularly receiving its usual amount from the left auricle. A progressive, alternating seesaw of hypertrophy and dilation of the left ventricular wall is thus set up which causes in time the greatest known size of heart, or *cor bovinum.*

Sooner or later, when dilation predominates nearly permanently over the hypertrophy, a relative mitral insufficiency develops, with its train of consequences already described. The presence of the double murmur in the arteries (Duroziez's sign) is, as intimated by the conditions explained above, virtually pathognomonic of aortic insufficiency.

DIFFERENTIAL DIAGNOSIS.—The differentiation of the *mitral obstructive* and "Flint murmurs" has already been pointed out in connection with the preceding case.

The interpretation of a diastolic murmur at the base of the heart may be difficult in the absence of distinctive arterial signs and the evidences of left ventricular enlargement, as in the early cases. For instance, such a murmur may be *functional* or *anemic,* although, it is true, rarely so; it may be due to congenital *pulmonary regurgitation* (also rare), to *aneurism,* to *fatty* and *relaxed* (*dilated*) *aorta,* or to *adherent pericardium* (Cabot), and *myocarditis* or *exophthalmic goiter* (Musser). If the murmur is hemic, and transmitted downward from the cervical veins, instead of aortic regurgitation, pressure over the jugular bulb will obliterate it. The rare pulmonary insufficiency is unaccompanied with hypertrophy of the left ventricle, and cyanosis and childhood or adolescence, instead of pallor and advanced years, are associated with it. Aneurism has expansile pulsation, often tumor, upper sternal dulness, and also the characteristic bruit and pressure phenomena. Dilated aorta usually gives an area of dulness between the first and third ribs along the right sternal border.

Associated aortic or *mitral systolic murmurs* are not infrequent with aortic regurgitation.

Aortic systolic murmur may often be heard, due to accompanying stenosis or roughness of the aorta or valve-segments. This murmur is usually harsh and high pitched as contrasted with that of aortic insufficiency, which is diastolic; it is transmitted into the carotids with the same quality, but in coexistence with aortic regurgitation, the soft murmurs in the other arteries often associated with the latter are absent; the murmur is also heard over a larger part of the body of the heart, in most cases, and is accompanied with a systolic tremor or thrill; and lastly, the pulse-waves are not so high and quick, if at all, as when the insufficiency is uncomplicated. One source of error in inferring aortic stenosis due to pathologic changes at the orifice, as well as leakage, should be indicated; namely, that the systolic murmur may be the result of a "physiologic stenosis," or really the normally narrower aorta beyond a very widely dilated aortic orifice, which thus permits a large column of blood to be propelled into the smaller caliber with current disturbance.

A *systolic* murmur in the *mitral* area associated with the aortic diastolic is not at all rare, both being often heard distinctly close together because of the transmission of the latter. The mitral systolic is softer than the aortic systolic murmur; it is heard to the left in the axilla; the pulmonic sound is accentuated, and secondary hypertrophy and dilation of the right ventricle appear later. The mitral insufficiency may be due to a combined endocarditis, or to a relative insufficiency, as from dilation of the left ventricle from other causes. Still later, a tertiary relative insufficiency at the tricuspid orifice may develop, as evidenced by soft systolic murmur heard best over the lower third of the sternum, and separated from the area of mitral regurgitant murmur by a zone or interval of feeble or absent audibility.

PART II

THE ABDOMEN AND ITS PRINCIPAL ORGANS

SECTION V

GENERAL EXAMINATION OF THE ABDOMEN

CHAPTER XVI

TOPOGRAPHIC ANATOMY, REGIONS, AND METHODS OF ABDOMINAL EXAMINATION

ANATOMIC ZONES, REGIONS, AND LANDMARKS

FOR descriptive purposes and the proper location of abdominal physical signs, we use terms expressive of anatomic regions having natural points of reference and more or less arbitrarily drawn limiting lines. According to the old traditional method, the abdominal surface is divided into three zones by two horizontal lines or imaginary planes, the upper being called the *infra-* or *subcostal,* which passes across at the level of the lowest part of the thorax (tenth rib), and the lower the *bispinal,* connecting the anterior iliac spines, thus producing the *epigastric, mesogastric,* and *hypogastric zones.* Subdividing these three zones by two vertical lines or planes passing through the middle of Poupart's ligament on each side (corresponding practically to the projected nipple-lines), we have then nine regions or spaces, as follows:

Right hypochondriac.	Epigastric.	Left hypochondriac.
" lumbar.	Umbilical.	" lumbar.
" iliac.	Hypogastric.	" iliac.

The iliac regions are also called sometimes the right and left *inguinal.* The hypochondriac regions may be made to coincide more naturally by having their inner and lower boundaries represented by the curved lines of the inferior costal borders (Butler). The lumbar regions may be considered as prolonged around the sides to the middle of the back, and subdivided further by the axillary lines.

Another regional division, much more simple and satisfactory, is that of the *quadrants* devised by Ballance, and indorsed especially by Musser. The surface of the abdomen is thus divided by a median and a transverse umbilical line into right and left upper and right and left lower quadrants. "The *right upper quadrant* contains the right lobe of the liver, the gall-bladder, the hepatic flexure and part of the transverse colon, a portion of the pancreas, the pylorus, near the median line, and, deeper, the upper half of the kidney. The *left upper quadrant* contains the left lobe of the liver, the stomach, part of the transverse colon and the splenic flexure, the pancreas, the upper portion of the kidney, and the spleen. The *right lower quadrant* contains the cecum, the ascending colon, the appendix vermiformis, the right tube and ovary, portions of the bladder and uterus, and above, at the end of full inspiration, the lower portion of the

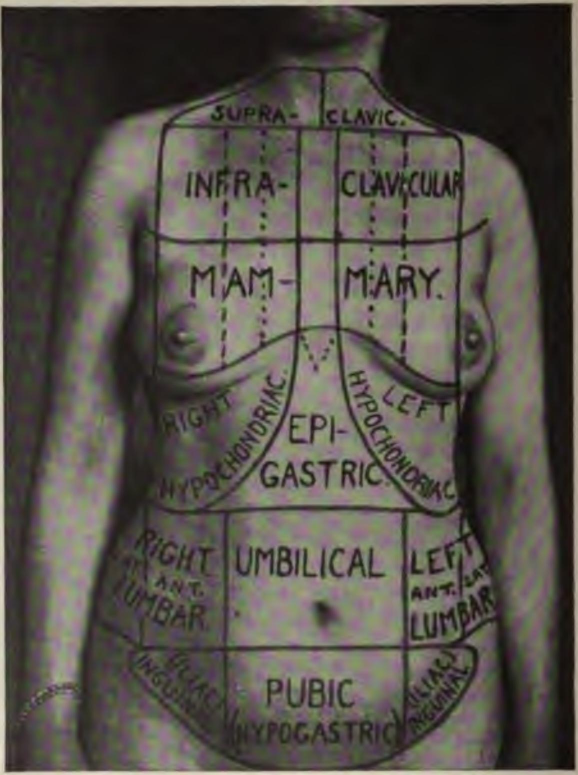


FIG. 75.—SHOWING NINE TOPOGRAPHICAL AREAS OF ABDOMEN. (After Joessel. Redrawn and modified.) (Butler.)

kidney. The *left lower quadrant* contains the corresponding tube, ovary, and portions of the bladder and uterus, the descending colon, and the sigmoid flexure. It does not usually contain the lower portion of the left kidney, which is one-half inch or more higher than the right (Holden). About the center, and extending to the periphery on all sides, are the small and large intestines."

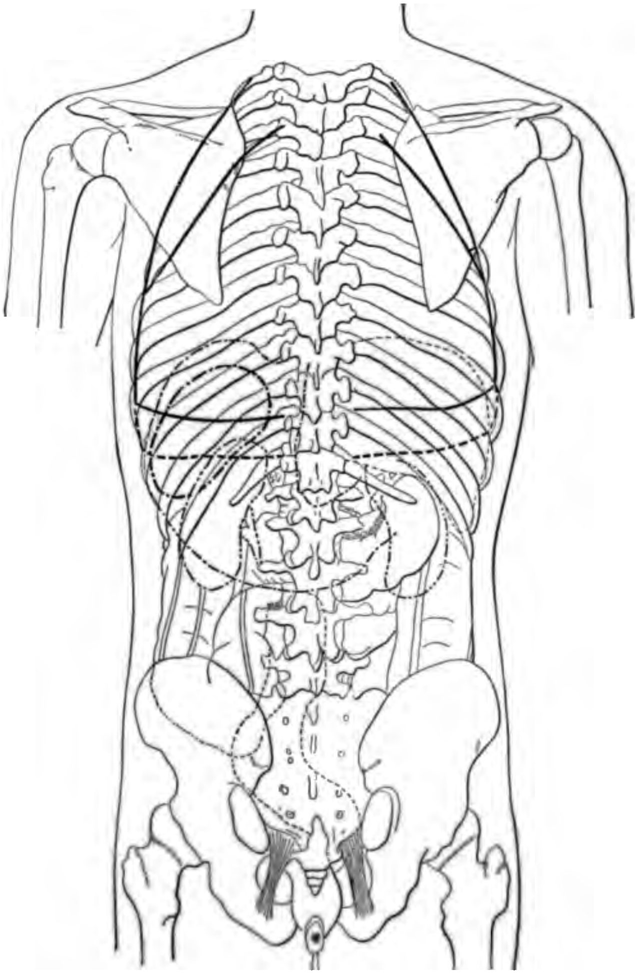


FIG. 76.—VISCERA OF THE THORAX AND ABDOMEN AS SEEN FROM BEHIND. (After Luschka: from Musser.)

Similarly, I have found it desirable at times to take a middle course, and divide the abdominal circle into *sextants*, approximately, by using three lines, one horizontal and two diagonal passing through the navel, the latter practically connecting the axillary regions with the middle of Poupart's ligament on opposite sides (see Plate IX).

The anatomic *landmarks* are useful in relational designation, and as measuring points for locating physical signs. The most reliable marks are, of course, the immovable or bony ones, especially the anterior superior iliac spines. The ensiform appendix, down-curving rib borders, and pubic symphysis are also easily distinguishable.

The other principal landmarks are the *linea alba, lineæ semilunares, lineæ transversæ,* and the *umbilicus.* Any portion of the horizontal or vertical line drawn through the last named may be used as a measuring basis to locate more accurately the results of physical examination. In determining the lower border of the liver, or of the spleen, when either organ is enlarged, we often speak in terms of one (¾ in.) or more fingerbreadths below the costal margins.

The *topographic anatomy* of the *abdominal aorta* needs a special reference. The aorta extends from a point midway between the upper border of the sternum and the umbilicus, along the left of the linea alba to its bifurcation, about ¾ in. to the left of and below the umbilicus, about opposite the body of the fourth lumbar vertebra.

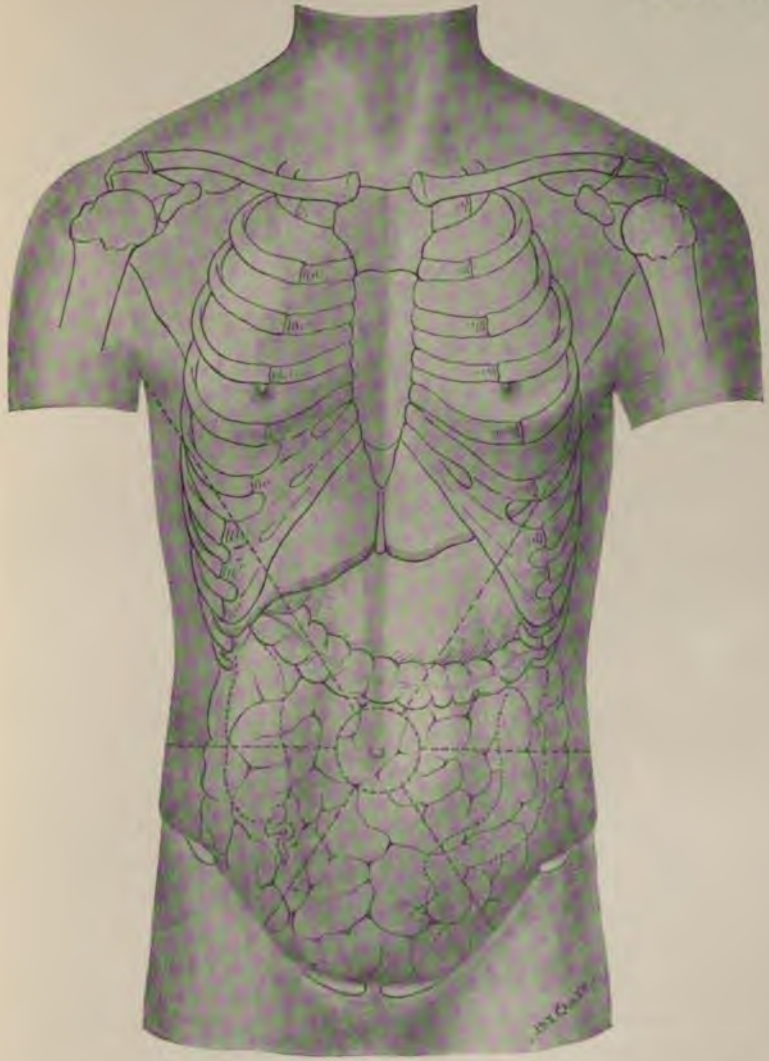
METHODS OF ABDOMINAL EXAMINATION

The relative importance and value of the usual four methods of physical examination as applied to the thorax are not the same as applied to the abdomen; their order is virtually reversed as regards fulness and exactness of results. Auscultation is rarely used, and affords little of value; percussion may often be of direct and confirmatory value, but is seldom practised under favorable conditions; inspection and palpation elicit the more reliable signs, but even with these methods, anatomic, functional, and complicating restrictions sometimes perplex, and prevent the determination of precise data.

GENERAL INSPECTION

Technic.—The patient should lie upon a regular examining table or chair, such as gynecologists use, although very often the examination must be made while the patient is in bed. If the latter, whenever possible a narrow bed with firm but comfortable mattress should be chosen, and the head supported upon a pillow so as to elevate the chest a little. Care should be taken that the cervical, dorsal, and

PLATE IX



ABDOMEN DIVIDED INTO SEXTANTS FOR THE LOCALIZATION OF PHYSICAL SIGNS.

lumbar spine are kept straight in the recumbent position. Sometimes, as in observing the shape of the abdomen with a view to estimating how much it may be influenced and how, by movable organs, effusions, and growths, the erect posture of the patient is necessary. The abdomen must be exposed completely, from the lower borders of the breasts to the pubes. It must be viewed, also, with a good, somewhat slanting light, such as falls upon the foot of the bed from a window directly opposite its head, and the examiner should look from both sides and at various angles, so as not to miss slight exaggeration of surface inequality and movement which shadows may cause. Heavy bedclothing should not be rolled down so as to dim the light over the lower part of the belly, but folded down far enough so that there is nothing more than the thickness of a sheet or thin blanket to cover the genitals and lower extremities.

The Normal Abdomen.—Its contour in the average individual of medium build shows a shallow arching, with the convexity most marked at or near the umbilicus if the person is lying down, or a trifle below that point if standing. After a full meal the upper zone, especially between the costal borders, may exhibit more than usual prominence. In infants the abdomen is relatively larger and rounder; also in women than in men; with more subcutaneous and omental fat. In multiparous women, and in those who have worn tightly fitting corsets, naturally the lower zone of the abdomen is more or less relaxed and pendulous—toward the flanks while recumbent, and overhanging the pubes while standing erect.

In *inspecting the abdomen for pathologic signs,* the following points are to be noted: (1) The size, shape, and general contour; (2) the surface appearances of the belly walls; (3) the respiratory and peristaltic movements; (4) local bulgings or depressions.

(1) SIZE, SHAPE, AND CONTOUR OF THE BELLY.—These may be considered together. *General enlargement* of the abdomen is usually *uniform* and *symmetrical.* It may be due to (*a*) fatty or edematous thickness of the abdominal walls; (*b*) usually to ascitic distention (dropsy of the peritoneum); (*c*) to gaseous distention of the intestines (tympanites), as from peritonitis, and in typhoid fever; (*d*) and to enormous organic or neoplastic enlargements crowding the abdominal cavity, such as massive uterine fibroid, ovarian cyst of extreme size, big sarcoma, great hepatic or splenic enlargement, etc. In cases where the enlargement alters its shape slightly with change of posture, as increase in fulness of the flanks in the recumbent position, and more conical protrusion forward, with the navel at or

a trifle above the apex in the erect position, ascitic fluid is commonly present. Gaseous distention of the bowels does not cause change of abdominal shape on changing the patient's posture. On very close inspection, the detection of slight asymmetry in an apparently uniformly enlarged belly justifies the presumption of tumor.

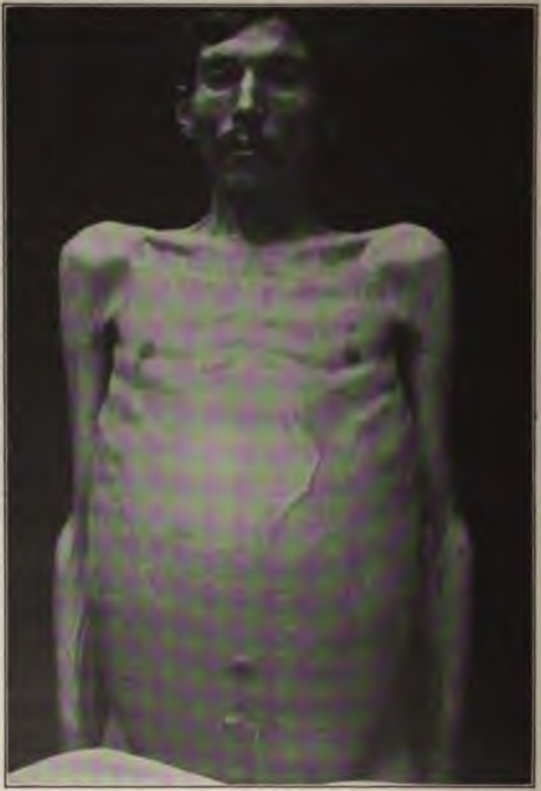


FIG. 77.—GENERAL ENLARGEMENT OF THE ABDOMEN FROM ABDOMINAL DROPSY (ASCITES). Note the turgid, tortuous vein.

(2) THE ABDOMINAL WALLS.—The various objective signs of the skin are concerned mostly in connection with general medical rather than with physical diagnosis and the grosser signs also subject to the other methods of examination. Thus, of the *color of the skin*, it need only be remarked here that its pallor or slightly dusky hue, with a tense, smooth, somewhat glazed or sheened appearance, is quite characteristic of ascites. If at the same time a number of *enlarged and tortuous veins* radiating about the navel (*caput Medusæ*) are seen,

and if the *umbilicus* levels up or protrudes, the presence of fluid in the peritoneal sac, due usually to atrophic cirrhosis of the liver, is undoubted. *Striæ* or *lineæ albicantes* may also be seen, bluish or slightly reddish at first, as accompaniments of prolonged skin-stretching due to ascites as well as to pregnancy.

A *retraction* or sinking-in of the abdominal walls is usually associated with the appearances of great emaciation and of a parchment-like hardness and color of the skin. It may be due to a tuberculous enteritis or peritonitis, a basilar meningitis (usually tuberculous), or to cancer, principally of the gastro-intestinal tract or peritoneum.

(3) MOVEMENTS.—These may be *respiratory, peristaltic* (gastric and intestinal), and *circulatory.*

Respiratory abdominal movements are naturally less marked in women than in men, and more marked in infants than in adults. *Increased* respiratory movements are compensatory to diseases of the upper thorax which interfere with free and full expansion. *Diminished* movements indicate diseases below the diaphragm accompanied with peritoneal pain, general or local, distention of the abdomen by large tumor, etc., or paralysis of the diaphragm.

Abnormal peristaltic movements suggest chronic pyloric or intestinal obstruction or stenosis, often due to cancer. However, in certain thin persons with relaxed abdominal walls, comparatively healthy but nervous, one may not infrequently witness slowly undulating vermicular movements passing beneath the walls, particularly after some such surface excitant as a tapping or filliping with the finger, pressing with a cold hand, or brushing lightly with the end of a towel wrung out of cold water.

Visible movements in the left upper quadrant are presumably caused by dilation of the stomach, the condition pointing to obstruction of the pylorus. Waves creeping around the umbilical region indicate swollen small intestines, with the obstruction at or near the ileo-cecal valve; those following a circumferential route along the colon, and in a reverse direction from the gastric ones (left to right), suggest obstructive disease at or near the sigmoid flexure.

Vascular movements may be communicated to the abdominal wall directly or indirectly by the aorta, causing epigastric or umbilical pulsations. The latter are often seen in nervous, tense, thin subjects, especially women. Or they may point to aneurism, or to a carcinomatous or sarcomatous growth of the pylorus, intestine, or mesenteric glands overlying the aorta and transmitting the pulsations. *Vascular pulsation of the liver* is sometimes visible in the right upper quadrant synchronous with the apex-beat, and associated with tricuspid insufficiency due to various causes.

(4) Local Abdominal Enlargements.—These are very important, although more amenable to palpation than to inspection, and will be more fully discussed under that head. Circumscribed distentions of the belly may occur in any region; they may be nodular and the size of a nut, or as large and irregular in outline as a fist or pumpkin; they may be fixed or movable, according as they may be affected by respiration and posture; they may appear to be a part of or attached to viscera, or independent neoplasms, or inflammatory exudations. Visible enlargements in the *right upper sextant* are usually hepatic or cholecystic, and movable with respiration; rarely, a movable right kidney may also be seen to cause slight bulging of the abdominal wall. In the *middle upper sextant* the most common protrusions are due to pyloric cancer; less common are pancreatic growths and aneurism. In the *left upper sextant* splenic, gastric, and colic tumors are most often found. Cecal tumors and appendiceal abscesses, as well as ovarian enlargements, cause local swellings in the *right lower sextant* most frequently, although I have seen displaced kidney in this region several times. The causes of visible bulging in the *middle lower sextant* may be tumors of the small bowel, mesenteric growths, uterine and vesical enlargements. In the *left lower sextant,* sigmoid tumors and fecal impactions, ovarian and omental growths appear.

Local depressions are rare, but may occur in the right or middle lower sextants from chronic peritonitis with circumscribed adhesions.

PALPATION

Knowledge of the value and mode of palpation alone will not suffice for a certain deficiency in knack, which cannot be described; but a good basis for acquiring skill in practising this method of obtaining and interpreting physical signs should be gotten by careful attention to a few leading facts and guiding principles.

The *position of the patient* is of first importance, the object being to have the abdominal muscles as relaxed as possible. Hence, the recumbent posture, with the head and shoulders slightly elevated, sometimes with the knees drawn up, but not always, as many patients relax less than when the legs are extended, and their elevation may be in the way of the examiner's arms, or throw a shadow over the exposed abdomen which is not satisfactory, as the eyes work with the hands during manipulation. Having the patient breathe quietly with the lips slightly parted aids in softening the belly walls automatically and psychically by diverting his attention. In nervous, timid, and ticklish patients it is often necessary, before going on with palpation, to divert the mind by engaging in conversation, feeling the pulse with the other hand, observing the tongue, palpating gently elsewhere than over the particular region of purpose, and so on. In urgent and extreme cases, anesthesia may be required.

The *hand of the examiner* should be warm, so as to avoid reflex contraction of the abdominal muscles. The first touch should be the placing of the whole hand upon the abdomen with light pressure, and then gradually passing it over the surface with circular movements and increasing pressure with the palmar aspect of the fingers, so as to accustom the patient to the palpation, and not resist the deeper and more detailed investigation to follow. Poking bruskly with the finger-tips in vertical fashion must consistently be avoided if the examiner wishes to retain the confidence of his patient. Therefore, also, painful and tender areas should be ascertained before touching, as nearly as possible, by heeding the patient's references, so that their exact location may be determined last, otherwise considerable tenseness and reflex irritability of the walls may ensue to restrict and confuse the doctor. Slightly deeper pressure and digital grasp is often afforded at the end of the expiratory movement of the belly wall, with apparently greater natural tolerance. Sometimes, when the resistance of the abdominal wall is especially marked, two hands may be employed, one on top of the other, with the finger-tips of the superposed hand resting back of the nails of the under hand, a reenforced pressure thus being maintained during several respirations while the lower hand is gaining impressions steadily. This method is often necessary in exploring the upper middle region of the belly, because of the contracted recti muscles and the central and lateral tendinous ridges bounding them longitudinally. Good relaxation may also be obtained in many intractable instances of abdominal rigidity by making the examination while the patient is nearly stretched out in a tub of hot water, the temperature of which has been raised gradually from 100° to about 110° or 115° F., the patient being immersed meanwhile. Further special points in palpation will be mentioned in connection with the conditions and organs to which they are applicable.

In palpating the abdomen we seek information on the following: (1) The wall itself; (2) general enlargement and retraction; (3)

local bulgings or depressions (rare); (4) pulsations, and movements due to respiration and peristalsis.

(1) **The Abdominal Walls.**—The *skin* may be smooth and feel doughy or putty-like, particularly at the sides of the belly, because of *edema*. The proportion of *fat* and the *thickness* of the walls may be estimated by grasping them upward. Relaxation and thinning of the walls are characteristic after pregnancy, dropsy, and in old age and during the progress of visceral carcinoma. The bellies of the recti muscles may manifest a general *rigidity* in nervous and ticklish individuals, in tetanus, muscular rheumatism, and peritonitis. Very often a *local contraction* of abdominal muscle is significant of local peritonitis, of appendicitis especially when the right rectus is so affected. These so-called muscular tumors differ from real growths in their variable resistance, since they relax and harden alternately, are not movable as most intra-abdominal tumors are, and give the impression of being superficial.

Other palpable irregularities may be *abscesses*, occasionally of tuberculous origin; *herniæ*, epigastric, umbilical; *neoplasms*, such as the lipomas or soft fatty tumors, or sarcomas; *separation of the recti muscles*, causing a soft projection in the median line, best demonstrated in bending the head and shoulders forward while lying on the back; *thickening, fixation, and retraction of the umbilicus*, suggestive of tuberculosis or cancer of the peritoneum or liver. The hernias may be recognized by their soft, air-cushion feel, with fine gurgling sensation on pressure, their reducibility, their increased tension and communicated impulse on coughing, and the tympanitic percussion note. Lipomas are soft, but feel fleshy, are more or less movable under the skin, and show their lobulated structure by causing tiny depressions or dimplings in the overlying skin when it is drawn firmly over the tumor.

(2) **General Enlargement of the Abdomen.**—This may be due in the first place to an EXCESS OF MURAL OR OMENTAL FAT, as in general *obesity*. If so, the enlargement of the belly is then found to be proportionate to the bulkiness of the extremities. A fat abdominal wall, however, makes it always difficult to palpate and discover coexistent tumors within the abdomen.

The intra-abdominal physical causes of general enlargement are principally three: an excessive quantity of *gas* within the stomach and bowels, *liquid* (usually a dropsical transudate) in the peritoneal sac, or a very large abdominal *tumor*.

If the enlargement is due to ACCUMULATION OF GAS within the intestines, producing the condition known as *meteorism or tympanites*, the abdomen is arched like a barrel, feels tense, although slightly resilient, and is accompanied with exaggerated thoracic and impeded cardiac action. The *causes* of moderate distention are often dietetic or gastro-intestinal; in children, usually due to the too free feeding of starchy preparations, and in adults to similar and sweet and fatty foods, such as oatmeal, hot breads, rich soups, fried meats and potatoes and sweet potatoes, beans, pastry, bananas, etc. There may be a congenital dilation of the colon with more or less permanent flatulence, or the dilation may be due to intestinal obstruction low down. I once saw an enormous abdominal enlargement in an aged minister, in whom the rectal tube could not be passed more than six or eight inches, and where the autopsy showed great distention upward and inward of the sigmoid loop, which was kinked and twisted (volvulus) at the pelvic rim. Typhoid fever, peritonitis, and hysteria may also give rise to severe gaseous distention of the bowel. In a few cases the general distention may be caused by *free gas* in the peritoneal cavity, the result of a perforated gastric or intestinal ulcer, or appendix, or of infection from the gas-forming bacillus. In such instances the lateral liver dulness is apt to disappear when the patient lies on the left side, the gas then being free to diffuse between the liver and the ribs; similarly with respect to the splenic dulness when the patient lies on the right side.

FLUID DISTENTION of the peritoneum is proven partly by the increased sense of resistance without the drumlike resilience, but mainly by the detection of *fluctuation*. This is elicited by applying one hand—feeling principally with the finger-pulps—to one side of the belly, while the other side is tapped gently but with quick impact by the palmar surfaces of the fingers of the other hand. If this is done near the supposed level of the fluid, the former hand receives the impression of the wave propagated by the percussion tap of the latter. Sometimes the lightest touch of a single finger suffices to develop fluctuation. If this sign is not demonstrable in the flanks, one may find it lower down and nearer the middle line. In stout individuals, who are at the same time apparently ascitic, the wave may actually be one of flabby fat simulating the liquid one. The possibility of error may be eliminated by having the patient or an assistant apply the ulnar edge of the hand longitudinally over the linea alba, which thus cuts off the superficial wave. The liquid fluctuation of ascites is also visible in its travel across the abdominal surface. If the amount of fluid in doubtful, undistended cases gives rise even to no local fluctuation in the sitting or recumbent posture, it may be elicited in the umbilical region while the patient assumes the knee-chest position.

The *causes of ascites* are principally cirrhosis of the liver, and the later stages of chronic cardiac dilation from valvular lesions, and renal disease with general dropsy. Less frequently occurring etiologic factors are interference with the portal circulation by a thrombus in the portal vein, or by pressure from without, as an abdominal tumor; interference with the general venous circulation by pulmonary emphysema (advanced), and chronic tuberculous and cancerous inflammation of the peritoneum.

Solid general distention of the abdomen from tumor may be inferred palpably by the firm, unyielding resistance to pressure, the more or less perceptible irregularity of outline, and sometimes bosselation or nodulation of surface, more marked lateral than anterior projection at times, and the absence of fluctuation.

Among the most common *organic and neoplastic causes* of general abdominal distention are the following: great enlargements of the liver (cancerous, amyloid, leukemic, malarial, etc.); spleen (malarial, leukemic, amyloid); kidney (sarcomatous, cystic); dilation and prolapse of the stomach (*gastrectasis, gastroptosis*); ovarian and parovarian cystoma and uterine fibroma; retroperitoneal and peritoneal lipomata and sarcomata; mesenteric disease, cancerous especially. Caution is always necessary not to mistake a large pregnancy for a tumor of pathologic origin.

General retraction of the abdomen is usually accompanied with marked sensation of resistance, thickness, of boardlike hardness sometimes. I have found it characteristic of chronic tuberculosis of the peritoneum and intestines; somewhat of general abdominal carcinosis, with a sort of bogginess to the touch; and also of chronic lead-poisoning with colic; in children with tuberculous meningitis, and in omental interstitial or cancerous thickening, particularly resistant in the upper half of the belly, and the mass not movable with respiration.

(3) **Local Enlargements.**—The detection of these is of the first importance in the palpation of the abdomen. The following points should be noticed: *Location* as to surface and region, *size, contour, mobility, consistency, relationship to adjacent structures and organs,* the presence of *pulsation* or *crepitation,* and *tenderness.* These features of local swellings are recognizable only by careful attention to and practise in the details of technic. It is considered best here to

indicate the main characteristics of the local swellings likely to be met with by classifying them regionally.

EPIGASTRIC REGION, OR MIDDLE UPPER SEXTANT.—Here as elsewhere it must be determined first whether the tumor is in the abdominal wall or in the abdominal cavity; and also whether, if in the abdominal wall, it is really a growth or simply a localized spasmodic contraction of a belly of the rectus muscle. If the latter, gradual relaxation with continued moderate pressure may be felt, and repeated and exaggerated contraction may be elicited by flicking the overlying skin, or by having the patient attempt to lift the head and shoulders from the dorsal position.

If the bulging is really a *tumor in the wall of the abdomen,* both may be partially or completely grasped and lifted, whereas an intra-abdominal tumor slips from under the seizure of the abdominal wall, and eludes the palpating fingers more readily than when the wall is not so manipulated. Mural swellings in this region may be ***abscesses*** or *fatty growths.*

Distention of the stomach from food or gas, or from permanent dilation due to cicatricial (healed gastric ulcer) or cancerous stenosis of the pylorus, may be noted here, although the latter condition is more commonly palpable lower down and more to the left. A *large parietal cancer,* such as the colloid variety, may also be felt in this region as a diffuse, firm mass; likewise a pyloric tumor.

Pancreatic cyst and *cancer* may be felt by deep palpation.

Liver enlargements, such as cancer nodules, hydatid cysts, abscess, gumma, and downward displacement of the left lobe, occur here. They are superficial and move with respiration.

Aortic aneurism is told by its round, firm outline and expansile pulsation.

The *transverse colon* may exhibit distention (soft, doughy sensation) or tumor.

The ***gall-bladder*** may be distended with pus or calculi (right side of epigastric region); if the latter, some ***crepitus*** may be felt.

The ***lesser peritoneal cavity*** may contain an effusion.

There may be ***enlarged mesenteric or retroperitoneal glands,*** from tuberculous, cancerous, or Hodgkin's disease. In these cases not infrequently aortic pulsation may be transmitted.

HEPATIC REGION, OR RIGHT UPPER SEXTANT.—***Liver enlargements*** of all forms are palpable here; for example, passive congestion, fatty, amyloid, malarial, cancerous, leukemic, syphilitic liver, hydatid disease, hypertrophic cirrhosis (biliary, or the first stage of the atrophic form), and abscess.

Distended gall-bladder may be due to hydrops, pus, concretions, cancer. The viscus is often pear-shaped, and may be swung in a short arc of circle whose radius is the length of the enlarged organ.

Subdiaphragmatic abscess may be the cause of a boggy bulging. A *perinephric abscess* likewise.

The *hepatic flexure* and adjacent portions of the *ascending and transverse* colon may be the seat of cancer or fecal impactions.

Movable kidney and *renal enlargements* palpable here may be hydronephrosis, pyonephrosis, sarcoma, cystic growth, rarely cancer of the suprarenals, or hypernephroma.

APPENDICEAL REGION, OR RIGHT LOWER SEXTANT.—In this very important area of modern medical and surgical attention, in the first place, conditions in the belly wall must be studied. Thus, *psoas abscess* due to tuberculous caries of the vertebræ may point here low down. *Inguinal hernia* is also of common occurrence.

Pericecal or periappendiceal abscess may be felt after the first two or three days of a severe acute appendicitis. There is an indistinct resistant sense of fluctuation. *Chronic appendicitis* may be palpated as a small, sausage-shaped tumor, or in those with thin, relaxed belly walls, as a cordlike thickening.

Fecal impaction in the cecum, with or without typhlitis, perityphlitis, or abscess, may be palpable here as a rounded, uneven, doughy, firm mass, with or without tenderness and external swelling. *Enteroliths* may also occur.

Cancer of the cecum and ascending colon causes a hard, irregular lump.

Intussusception is found more often in children as a firm tumor with the caliber of the bowel, associated with a straining to defecate. The tumor is usually felt nearer the navel than the other swellings of this region.

Retroperitoneal sarcoma may project in this area as a large mass.

Floating kidney is not at all infrequently palpable here.

The following *pelvic enlargements* may be palpable in this region: *ovarian tumor*; *tubal swellings*, as pus tubes of large size; *pelvic abscess*; *extra-uterine pregnancy*; *pelvic hematocele*. Tumors of the right ovary are usually firm when small and barely palpable. As they increase in size they may become somewhat elastic in feel, and since they are often cystic fluctuation may be elicited. The enlargements are nearer the median line than those of appendiceal or typhlitic origin. The other pelvic swellings also occur in the lower half of this region, nearer the median line than the perityphlitic tumors.

PELVIC OR PUBIC REGION, OR LOWER MIDDLE SEXTANT.—Swellings here may be due to ***distended bladder, fibroid*** or ***pregnant uterus, tuberculous peritonitis,*** and the pelvic enlargements mentioned in the previous paragraph. A long and *inflamed appendix* may sometimes be found in the right side of the pelvic region, and this area may project as a part of a diffuse, baggy bulging of the lower zone of the abdomen from *dilation of the stomach* and *gastroptosis.*

SIGMOID REGION, OR LEFT LOWER SEXTANT.—Near Poupart's ligament one may palpate *hernia, enlarged glands* (syphilis, cancer, Hodgkin's disease), and *psoas abscess. Carcinoma* of the *sigmoid flexure,* hard *fecal accumulations,* and *perisigmoidal abscess* may all be palpable here. Volvulus, and rarely a *displaced kidney* or *spleen,* may occur. In women, near the inner portion of this area the *ovarian tumors, broad-ligament cysts, hematomas,* and *intussusceptions* may be met with.

SPLENIC REGION, OR LEFT UPPER SEXTANT.—One may feel here an *enlarged* or *dislocated spleen;* a *mobile kidney;* a *gastric cancer* or *dilated stomach; renal tumors,* as *cystic kidney* and *perinephric abscess;* local *peritoneal effusions;* and *enteroliths* or *impacted feces* in the *splenic flexure* of the colon.

UMBILICAL, OR CENTRAL REGION.—*Umbilical hernia* is frequently perceived here, especially in young children. It is soft, and often feels like a little air-bladder with tissue-paper walls, which presses out against the finger with coughing and crying. A hard, cordlike adhesion, tender to the touch, and usually associated with colicky attacks, is too often overlooked. The attachment may be a *fetal remnant* or an *omental adhesion.*

Also palpable here may be the following: *dilated stomach,* large *pyloric cancer,* which has dragged the stomach downward; *floating kidney, spleen,* or *liver; prolapse* and *tumor of the colon;* the soft, pendent bulging of general *enteroptosis;* the hard, nodular, or lumpy mass of *retroperitoneal sarcoma; enlarged mesenteric glands* (cancer or tuberculosis); *shrinking and thickening of the omentum* from chronic peritonitis, cancerous or tuberculous growths; *hydatid liver* disease or *tumor* of the *gall-bladder;* and the expansile pulsations of saccular or spindle-shaped *aortic aneurism.*

(4) **Pulsations and Respiratory and Peristaltic Movements.**—The PULSATIONS OF THE AORTA have already been pointed out in connection with palpable aneurism in the regions (epigastric and umbilical)

in which it may occur. It is not uncommon, also, to palpate a marked aortic pulsation along the whole of its abdominal course in two classes of patients, namely, thin young women of neurotic and hysteric type, sometimes of the relaxed, tuberculous type, and those advanced in years, men and women, having tense, sclerotic aortæ.

The RESPIRATORY MOVEMENTS of the abdominal walls are exaggerated and jerky in nervous conditions, which may be temporary and disappear with diverting conversation and suggestion; they are diminished in painful diseases of the peritoneum of general or local character and origin, in lead-colic, large tumor masses, etc.

The slow VERMICULAR MOVEMENTS of peristalsis are seldom felt as easily as they are seen. They signify some chronic obstruction of the pylorus usually when in the upper zone of the abdomen, or a similar condition, as cancer of the intestine, when in the middle or lower zones.

PERCUSSION

The technic of abdominal percussion does not differ from that of thoracic except that, as a rule, very light strokes are advisable to prevent overlooking small areas of dulness because of the ease with which adjacent tympany may be elicited.

The NORMAL, GENERAL ABDOMINAL NOTE is, of course, TYMPANITIC. It varies, however, as to pitch and intensity, as well as clearness, in different regions, according to the underlying volume of viscus percussed, and its degree of emptiness of liquid or solid material. Thus, over the small intestines, around the umbilical area, the tympanicity is higher pitched and not as loud and clear as over the colon, nor over the latter as compared with the empty stomach. The degree of tension, also, affects the tympanitic sound—the greater the tension the higher the pitch; therefore, it is often difficult to differentiate stomach, large and small bowel by percussion near their anatomic borders, because of the numerous variations in size and tension which may affect one or more portions of the alimentary tract.

In the upper part of the epigastrium (middle upper sextant), owing to the presence of the left lobe of the liver, light percussion demonstrates slight dulness.

EXAGGERATED TYMPANITIC SOUND over the abdomen generally is associated with gaseous distention of the bowel, as in peritonitis and typhoid fever, or with enormous dilation and prolapse of the stomach, or with gas in the peritoneal cavity from a ruptured ulcer (typhoid, tuberculous, cancerous, gastric).

Ascites.—The commonest cause of more or less generalized dulness on percussion of the belly is fluid, usually dropsical, in the peritoneal sac. The location, size, and outline of the dulness depends, obviously, upon the quantity of the fluid present, and, since it gravitates to the lowest portions, upon the position of the patient. Hence, when the dorsal decubitus is assumed, dulness is elicited in the flanks if the effusion is sufficient to rise above the level of the heavy lateral muscles, and over a narrow zone at the lower part of the belly. The middle part gives tympanitic sound due to the floated bowels. In certain large individuals with large-calibered bowels and but moderate amount of liquid, although slight bulging in the flanks may be visible and local fluctuation be palpable, tympany instead of dulness may be demonstrable here nevertheless. When the patient lies upon either side, tympanitic sound is elicited over the flank of the upper side where in the recumbent posture dulness was found. In the standing position the dulness is confined principally to the lower half of the abdomen, in the median and parasternal lines reaching upward to the level of the navel, but seldom higher.

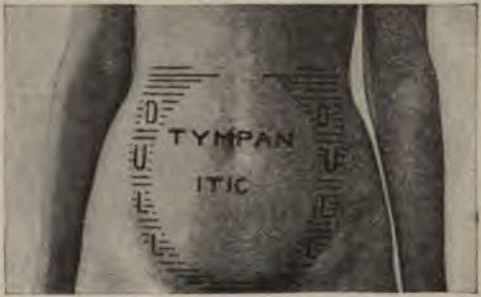


Fig. 78.—Showing the Central Tympanicity and Lateral Dulness of an Abdomen Containing Free Fluid. (Butler.)

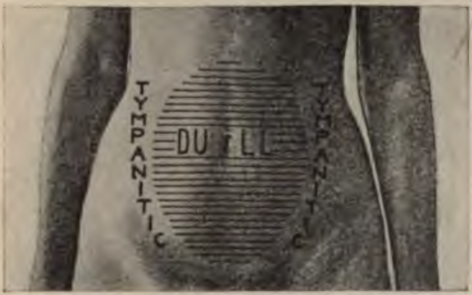


Fig. 79.—Showing Central Dulness and Lateral Tympanicity of Abdominal Cystic or Solid Tumors. (Butler.)

AUSCULTATION

Auscultation of the abdomen in general practically gives no information of any decided value. Any significance attaching to the sounds produced in the gastro-intestinal tract, and both the liver and spleen, will be narrated briefly in referring to these organs specifically.

In regard to the *peritoneum*, FRICTION SOUNDS or a soft, rustling CREPITUS may be heard whenever a plastic inflammatory exudate of extensive nature involves its surface where the respiratory movements may be communicated; hence is more often heard over the liver and spleen, as a *perihepatitis* or *perisplenitis*, or a *subphrenic peritonitis*. Creaking and crumpling sounds are sometimes audible in cases of old, adhesive peritonitis, often of pelvic origin, or fine crackles may be heard over the cecum in perityphlitis.

VASCULAR SOUNDS occur, such as the bruit of aortic aneurism (systolic), pressure murmurs (also systolic) due to growths encroaching upon the aorta, and the hemic murmurs of marked, as pernicious, anemia. A systolic bruit in the epigastric region has been observed, associated with expansile pulsation, in which the cause was undoubtedly a distended, displaced gall-bladder.

SECTION VI

THE PRINCIPAL SPECIAL ORGANS OF THE ABDOMEN

CHAPTER XVII

THE STOMACH AND INTESTINES

TOPOGRAPHIC ANATOMY OF THE STOMACH

THE stomach, pear- or flask-shaped, slopes a little obliquely downward from its larger (cardiac) or left end to the right or pyloric end, so that about five-sixths of the organ lies to the left of the median line of the body. The upper limit of the *fundus,* where it fits into the dome of the diaphragm, reaches to the fifth rib in the nipple-line, behind the apex of the heart. The fundus is adjacent to the spleen and left kidney, and is partly overlapped in front by the lower border of the left lung, the heart, and the left lobe of the liver.

The *cardiac orifice* lies back, or possibly ½ to 1 in. to the left, of the sternal junction of the seventh costal cartilage. It is at least 4 in. from the anterior surface of the body.

The *lesser curvature* bows downward in passing to the right from the cardiac, then ascends with a short curve to the pylorus. It also lies deeply, and is adjacent to the pancreas, above and behind.

The *pyloric orifice* is more superficial than the cardiac orifice, and also more movable. It lies in or slightly to the right of the middle, or even the right sternal line, from 1 to 2 in. below the tip of the ensiform cartilage. It is directly behind the liver.

The *greater curvature* has a gentle convexity downward and forward, connecting the pylorus with the fundus, and reaches the level of the infracostal line (connecting the tips of the tenth ribs). When the normal stomach is considerably distended, the greater curvature may reach as far as the navel. It is adjacent to the transverse colon below.

Thus the stomach occupies in part the left inframammary, th left hypochondriac region or left upper sextant, and the epigastri region or middle upper sextant. That portion of the stomach whic may be palpable forms a spherical triangular area bounded below b the greater curvature, to the right by the lower border of the live and to the left by the lower border of the costal cartilages.

The so-called *Traube's semilunar space* is the area of the lowe ribs which are in contact with or cover the parietal surface of th stomach. It is bounded by the liver and lung above, the spleen to th left, and the liver also (left lobe) to the right. Over this regio the true gastric tympanitic sound is elicited.

PHYSICAL EXAMINATION OF THE STOMACH

INSPECTION

The normal stomach has no visible signs.

Concerning the pathologic stomach, we aim to determine it (*a*) *position*; (*b*) *size*; (*c*) *movements*; (*d*) *tumors*.

Displacement and distention of the stomach may be considere together, as they are often associated conditions; a dilated (*gastrectasis*) stomach is usually at the same time prolapsed, especially if du to organic stricture of the pylorus; on the other hand, however, a prolapsed stomach (*gastroptosis*) may occur with such slight or moderate atonic dilation as not to be discoverable by physical examination. These cases may be indicated by more or less diffuse bulging in either of the left sextants, or in the umbilical region. The lower curvature may even be seen as a transverse shadow or line moving in the hypogastric or pelvic region with inspiration. The epigastric region, on the contrary, is relatively depressed. Of course, these signs are visible only in those individuals having comparatively thin abdominal walls. In marked gastroptosis a groove may be seen extending from the navel to the ribs which represents the lesser curvature of the stomach. These phenomena may all be better visible with the patient in the semirecumbent position, and the abdomen viewed from behind while the light falls directly upon it, although it should be carefully observed from in front and at various side angles as well.

The *respiratory movements* of the lower curvature of the stomach are generally seen only when the stomach is tensely dilated, either from the gas of fermentation or of artificial distention (see under Percussion). *Peristaltic movements* are also better visible under like conditions of the gastric walls. They may be seen to pass as slow waves from left to right, sometimes excitable by finger-tapping; if the pylorus is low down because of a weighty cancerous growth there, the waves show a distinctly downward course as well. Benign stenosis of the pylorus from cicatrices of healed gastric ulcer, adhesions, and simple thickening, may likewise be signified by the detection of peristaltic gastric waves.

Tumors of the stomach are not visible unless large, as in advanced cases of scirrhous cancer of the pylorus in emaciated persons, where the growth may appear well below the left lobe of the liver. Such neoplasms are not subject to respiratory mobility—a differential point in relation to tumors of the liver and gall-bladder.

PALPATION

Palpation of the stomach determines with fuller results the visible indications. The normal stomach cannot be palpated unless, in individual cases, it is greatly distended with a heavy meal, or from gas. Slight epigastric tenderness on pressure is common to most normal persons.

Tenderness is abnormally increased in palpating the stomach affected with acute or chronic inflammation (*gastritis*), when it is rather diffuse; in *gastric ulcer*, when it is sharply localized; and in *cancer*, when it is indefinite or circumscribed to the tumor area.

Displacement and *dilation* of the stomach cannot be palpated by its boundaries, except in rare instances in which the greater curvature may be felt assisted by the guiding of vision. The dilated stomach gives a soft, baggy feel below the borders of the left ribs, often as far as the umbilicus. A splashing or succussion sensation may be elicited when gas and liquid are present.

Increased resistance of the gastric walls means hypertrophic thickening of the muscular coat, which is usually associated with general distention of the organ. Firm and moderately diffuse resistance just below the rib borders may signify a large colloid cancer of the stomach walls. This must not be confounded with a tense belly of the left rectus muscle.

Peristaltic waves which can be felt as well as seen are important because of their indication of hypertrophic dilation of the stomach, secondary to some pyloric obstruction, and often giving a fair indication of the size of the stomach. Usually the waves undulate from left to right, but in certain extreme cases of stenosis they are reversed (or *antiperistalsis*). Kussmaul describes in the phrase "peristaltic unrest," a palpable peristalsis in neurotic individuals without gastric dilation.

Tumors of the stomach, especially the pylorus, cannot be felt unless the latter is pushed down and sufficiently parietal. *Carcinoma* is generally found as a dense, irregular mass, a little to the right of the median line, between the xiphoid and the navel, although in a few instances I have palpated it a little below the latter point. The cancer may be slightly movable with respiration, especially when bound to the liver by adhesions, and when low down is quite movable with the hand, and with change of posture. A diffuse thickening of the stomach wall in the left parasternal line just below the borders of the ribs may be due to a perigastric deposit as a consequence of a perforating gastric ulcer. Sometimes a pancreatic growth may be mistaken for a gastric cancer, and *vice versa.*

PERCUSSION

Percussion of the stomach must be done with the greatest care in technic, otherwise one may be easily misled both by intragastric and adjacent conditions. Normally, gastric tympany is limited to Traube's half-moon-shaped space, where it is parietal, although a modified or relative gastric tympany may be elicited beyond these borders. Gentle percussion suffices to discover it. The tympany is frequently high in pitch, although lower than that obtained over the intestines, generally; nevertheless, the tympany over the colon adjacent to the stomach is often indistinguishable from that over the latter, indeed, sometimes because the colon is so distended as to overlap the stomach. In order to separate gastric from colonic tympany it may be necessary to distend the stomach by the same means usually employed to ascertain its size in suspected cases of dilation (see below). Under other circumstances the amount of gastric tympany obtainable will depend greatly upon the condition of the gastric contents—the presence and relative quantities of gas and liquid or solid matter; upon the extent of overlapping lung and liver, either of which may be enlarged, and upon pleural effusion or splenic tumor.

Therefore, while the *upper border* of the stomach lies about on a level with the fifth to the sixth rib in the parasternal and nipple lines, and at the seventh or eighth rib in the anterior axillary line, one must really be content with defining the lung-stomach and the lung-liver boundaries, by noting in the first instance where tympanitic resonance (with light percussion stroke) ends and tympanitic clearness begins, and in the second, where clearness passes into tympanitic dulness or muffled tympany.

The *outer* or *left limit* of gastric tympany is determined by percussing to the left until splenic dulness is noticed near the lower borders of the ribs in the axillary lines.

The position of the *lower border* or greater curvature is most surely and accurately determined by artificially distending the stomach either with gas or water, preferably the latter, sometimes both; in any case, the object being to separate gastric from colonic tympany, the differences in pitch being usually so slight. Ziemssen's method consists in inflating the stomach with carbonic-acid gas, by giving the patient a heaping teaspoonful of sodium bicarbonate, dissolved in water, followed immediately afterward by a similar solution of tartaric acid. *If* the evolution of gas is sufficient to distend the stomach to its full size, but not so violent and abundant as to cause a sense of oppression or collapse from cardiac embarrassment, then this procedure may be deemed successful; but as, after the administration of these substances, the quantitative chemical result is beyond our control, the method is too uncertain and variable, and may be dangerous.

Therefore, a better method of distention, and a safer, is to pump air through a stomach tube, introduced previously, to which is attached an ordinary hand-bulb syringe; discomfort may be relieved at once by allowing the air to escape. The air should be forced into the stomach at first quickly and vigorously, so as to cause reflex closure of the pylorus, and thus prevent distention of the bowel. By this means the stomach may be outlined by inspection and palpation, as well as by percussion of the area of gastric tympany.

Still another method of determining the size and position of the stomach by its greater curvature is first to note a general tympany below the left ribs, in which gastric tympany is inseparable from the colonic, and then, while the patient is standing, have him drink freely of water, after which percussion will develop dulness over a zone representative, of course, of the lower limits of the stomach or region to which the water has naturally gravitated.

Auscultatory percussion is decidedly more accurate and satisfactory than simple percussion for outlining the area of gastric tympany, by determining the adjacent lung, liver, spleen, and colon borders, whether the stomach be normal in size and distention, or inflated, or partially filled with liquid. Concerning the last-named test, it is well to note the results after changes of posture, the patient having previously drunk one or two tumblerfuls of water. The dulness obtained while the patient is erect, where before tympany was elicited, indicates that the latter is gastric and not colonic; and when the patient is then placed in the recumbent position, tympany replacing the water dulness again points to its gastric origin, thus determining, by the location and limits of such signs, the position and extent of the stomach area.

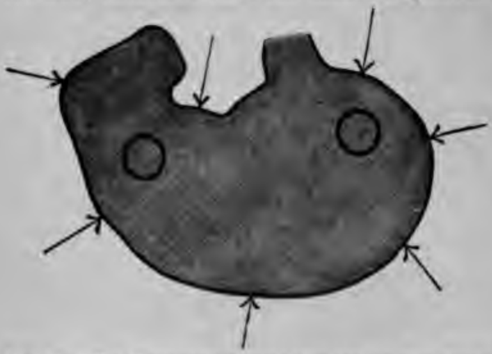


FIG. 80.—TO DETERMINE THE OUTLINE OF THE STOMACH BY AUSCULTATORY PERCUSSION. The stethoscope may be placed at either of the points indicated by the circles during the first percussion. During the repercussion it should be shifted to the other point. Arrows show lines along which percussion should be conducted. (Butler.)

In the *technic* of auscultatory percussion, a mere fillip or glancing stroke of the finger is all that is necessary in approaching the borders of the viscus, while the chest-piece of the stethoscope is held over the body or near the pylorus of the organ. The individual quality and pitch of the gastric note is perceived first by listening near the angle between the ensiform appendix and the left costal margin. Stroking percussion is then practised from all sides toward the stomach area until this same note is successively observed, which thus determines the boundaries of the stomach.

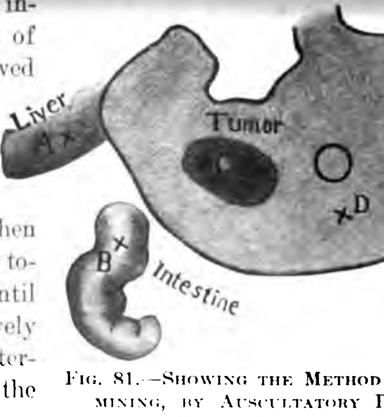


FIG. 81.—SHOWING THE METHOD OF DETERMINING, BY AUSCULTATORY PERCUSSION, THAT A TUMOR BELONGS TO THE STOMACH RATHER THAN TO THE LIVER OR INTESTINE. If the tumor involves the wall of the stomach the percussion notes over points *A*, *B*, *C*, and *D* differ, but *C* resembles *D* much more nearly than *A* or *B* resemble *D*. (Butler.)

Tumors of the stomach may also be discovered by this method.

Enlargement of the area of gastric tympany is seen in cases of dilation and downward displacement, as from pyloric tumor; extrinsically, because of cirrhotic atrophy of the liver, retraction of the left lung from fibroid phthisis, frequent pregnancies, etc.

The percussion-area is *diminished* by such external causes as encroaching enlargements of the liver and spleen, left-sided pleural effusion, pneumothorax, emphysema, pericardial effusion, cardiac hypertrophy.

Actual *diminution in the size* of the stomach is indicated if the greater curvature is observed to be higher than a level of 1½ to 2 in. above the navel.

AUSCULTATION

Very little of diagnostic value is obtained by this method of examining the stomach. In health, a sound is heard at the cardiac orifice of the stomach while liquid is passing down the esophagus through it, and there is normal rhythmic gushing or splashing sounds, which may be heard over the stomach, containing air and water only, when the body is shaken sharply. In health, also, the "deglutition murmur" just referred to is often followed by a squirting sound, some three to seven seconds later, as the liquid passes into the stomach at the cardiac orifice; this is best heard while listening at the left xiphocostal angle. A cessation or marked postponement of the gurgle or spurt at this point would indicate stenosis of the cardiac orifice, possibly a weakness of the swallowing muscles.

Splashing or *succussion sounds*, which may be elicited by a bed-patient simply changing position, or by a sudden, lateral movement of the body, or by the examiner pressing rapidly with the fingers of both hands alternately, with a plunging or gently poking motion. This is done between the level of the navel and the left costal border. If this splashing can be detected more than three hours after a meal of light character, or six hours after a heavy meal, persistently, it is evidence of *atony* or loss of motor power of the stomach, or of dilation and relaxation of the stomach, especially if the sounds are audible below the transverse umbilical line. Artificial inflation of the stomach will enable one to discover this phenomenon still more readily.

Bubbling, *gurgling*, and *clinking sounds* may be heard, often influenced by the respiratory movements while auscultating the left lung below the fourth rib or interspace. They are also significant of gastric dilation, with gaseous distention and some liquid.

Fizzing sounds indicate active fermentation going on, or the antiperistaltic efforts of the irritable stomach of nervous dyspepsia to eruct air or gas.

The heart sounds may assume a metallic, resonant quality in their transmission through a dilated stomach over which one is listening.

TOPOGRAPHIC ANATOMY OF THE INTESTINES

The *topographic anatomy* is best shown in Plate IX. It is to be remembered that the *small intestine* occupies principally the middle portion of the abdomen below the waist line.

The *large intestine* or *colon* takes up the periphery of the abdomen, from the cecum and appendiceal region to the hepatic flexure, near the right costal border, thence horizontally, with a slight curve downward (transverse colon) to the splenic flexure, passing thus above the umbilical level, to end in the sigmoid flexure lower down, near the left Poupart's ligament. The cecum is more superficially seated than the upper portion of the ascending colon.

The *appendix vermiformis* lies, at its root, at the middle of a line drawn from the anterior superior iliac spine to the navel, near the right edge of the rectus muscle.

PHYSICAL EXAMINATION OF THE INTESTINES

INSPECTION

Most of the general and local enlargements of the bowels have already been pointed out in the preceding chapter. Next to tympanites, the increased peristalsis or vermicular movement occurring above an intestinal obstruction is most important. Obviously, the less the area of distention and vermicular movement the nearer to the duodenum must be the stenosis. If the colon alone is distended with gas, its outline may be determined by its long, sausagelike swellings in the flanks.

PALPATION

Doughy masses of fecal accumulation are most often felt in constipated women with thin abdominal walls, especially over the cecum and ascending colon, sometimes over the sigmoid flexure. These lumps are also to be differentiated from tumors of the intestine by their disappearance after purgation or enemata. Rarely, *enteroliths* or bowel calculi may be felt.

Gurgling in the right iliac region is often felt in typhoid fever, but may be detected also in the normal individual at times, when the cecum is more or less filled with gas, and irritability of the intestinal motor function is present.

Peristaltic motion of a portion of intestine is palpable above a stenosis, and if the obstruction be chronic, this region of the bowel is moderately hypertrophied and resistant with distention. A malignant thickening and stricture of the colon is most likely to be found at one of the flexures or at the cecum.

Appendiceal thickening, swelling, and *abscess,* with hardening of the overlying and reflexly protecting rectus muscle, are characteristically palpable in this region.

Tumors of the colon are usually cancerous, and may be felt most frequently either in the cecum or at the sigmoid flexure. They are hard, often nodular and irregular in outline, with oblong masses of impacted feces above them. Intestinal neoplasms are rather mobile within the hand grasp, but fixed as to location relative to the other parts.

In *chronic tuberculosis of the bowel* one feels its coils as firm, cordlike, tangled masses through the thin but boardy abdominal walls.

PERCUSSION

Normally, the bowel sound is tympanitic, slightly higher pitched centrally over the bunch of small intestine than peripherally over the colon. However, as the conditions of volume and tension and content of the bowels are so variable, both locally and generally, absolute dependence can seldom be placed upon the percutory results. Light percussion strokes are best. Auscultatory percussion is more reliable in differentiating large from small intestine.

Moderate increase in gaseous distention of the bowel produces louder and lower-pitched tympany than normal, and over a greater extent of the abdominal surface, encroaching upon the hepatic and splenic dulness. In cases of *extreme distention,* as in severe peritonitis, the note may lose its distinctive tympanitic quality, and become more like the harder, "band-box" sound of the hyperresonance of emphysema.

To *outline the large intestine* by ordinary percussion the colon may be filled with water by high, postural enemata, and the dulness thus noted. On the other hand, by auscultatory percussion the colon may be differentiated by ballooning it with air pumped into it through a rectal tube. This may be useful where congenital or acquired prolapse and flexure are suspected from obstructive symptoms.

Areas of dulness over the intestinal region, or of dull or muffle tympanitic sound, are indicative of *tumors* or fecal impactions o accumulations, the latter being soon inferable if they are movabl under catharsis or enteroclysis. Cancer or malignant stricture of th colon is more apt to occur at the anatomic flexures, and the small dull, or relatively dull, area is characteristically separated from a distinct tympany due to the distention above and adjoining the stricture. Stengel claims that a tumor may be said to be referable to the wall of the previously ascertained colon when, by auscultatory percussion, the stethoscope being placed near the tumor and stroking percussion being made over and toward the tumor from all directions, the note over the tumor resembles that over the adjacent colon more nearly than the note over the small intestine resembles the latter. To discover the dulness over an intestinal tumor requires deep pressure with the pleximeter finger.

Chronic thickening of the cecum and appendix from recurrent attacks of appendicitis may be indicated by the persistence of moderate dulness here associated with increased sense of resistance (not due to muscular rigidity). A chronic appendiceal abscess may also be the cause of circumscribed dulness in this region.

AUSCULTATION

Certain rumbling, fine gurgling, and popping sounds occur rhythmically over the intestines in health. Intestinal noises, or *borborygmi* (rumblings), or cooing, or splashing sounds, when marked and persistent, are all suggestive of active or exaggerated peristalsis, with the passage of gas and fluid or semifluid matters through the loops and knuckles of gut. Borborygmi are usually associated with flatulent dyspeptic conditions, such as gastro-intestinal catarrh, especially in nervous and hysterical women and those who, having borne several children, have lax abdominal walls.

Loud *cooing sounds*, if associated with symptoms of intestinal obstruction, may have diagnostic value, being due to the difficult passage of gas and liquid through the narrowed part of the bowel; otherwise, the sounds may be heard occasionally in health. *Gurgling* in the ileo-cecal region is often heard in typhoid fever; it is developed or increased best by simultaneous palpation. *Splashing*, audible over the cecum, indicates a dilated condition of this portion of the large intestine.

CHAPTER XVIII

THE LIVER, SPLEEN, AND KIDNEYS

TOPOGRAPHIC ANATOMY OF THE LIVER

The liver being wedge-shaped, so that about three-fourths of it, or the thicker portion, lies on the right side of the body, thus occupies topographically the right upper sextant, or the right hypochondriac region, largely. Its thinner portion extends into the epigastric or upper middle sextant, its extreme left end reaching to the left parasternal line. The *upper surface* of the liver fits snugly into the diaphragm, its dome being covered by the concavity of the right lung; this highest point of the right lobe reaches the level of the fourth rib (lower border) or interspace; at its left end, the highest hepatic point is at the fifth interspace. The *lower surface* is in contact with the stomach, transverse colon, duodenum, and right kidney. The *anterior, lateral, and posterior surfaces* are in direct relation, below the lung limits, with the right lower ribs and the abdominal wall.

The sharp *antero-inferior edge* passes obliquely upward and to the left from the lower border of the ribs, with which it practically coincides, crossing between the ninth and tenth right costal cartilages; thence across the epigastrium, about one-third the distance between the xiphoid and umbilicus; thence across the eighth left costal cartilage to its left end below the heart. So that the greatest vertical extent of uncovered or parietal liver is practically in the right midclavicular line. In the midaxillary and scapular lines, the lower border extends along the eleventh rib.

The vertical extent of parietal liver thus tapers toward the spine, being three interspaces wide in the axillary line (eighth to tenth inclusive), and but one interspace wide in the scapular line (tenth to eleventh), as well as to the left across the epigastric region. The interlobar notch is virtually in the median line.

The position of the liver is affected by respiration; deep inspiration depresses it palpably. Also, when the patient lies upon the left side, the left lobe of the liver rises higher and the right extends lower, the suspensory ligament serving as an axis.

Age is a factor, the liver being proportionately larger in children, so that its lower border projects about an inch below the edge of the ribs in the nipple and midaxillary lines. In old people, on the contrary, due to contraction and atrophy of the parenchyma, the liver has diminished in size so that its lower border lies about one inch above the rib edge.

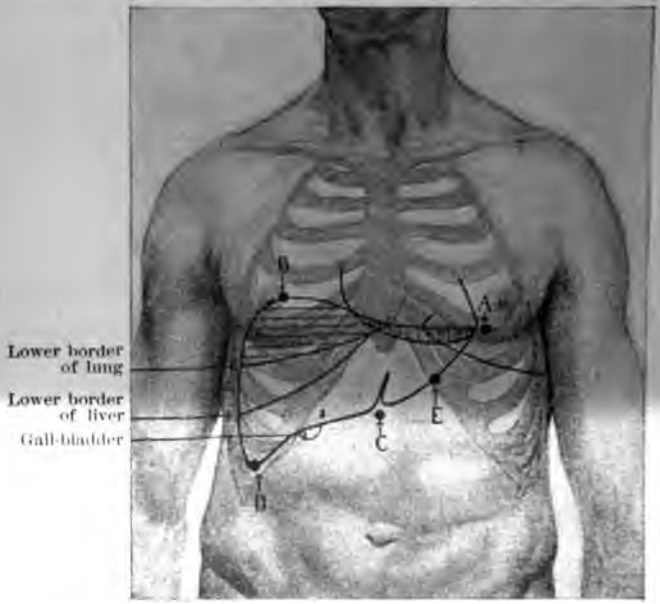


FIG. 82.—SHOWING THE POINTS WHICH DETERMINE THE SIZE AND POSITION OF THE NORMAL LIVER. Horizontal shading = portion of liver overlapped by lung; vertical shading = portion of liver overlapped by heart. (Butler.)

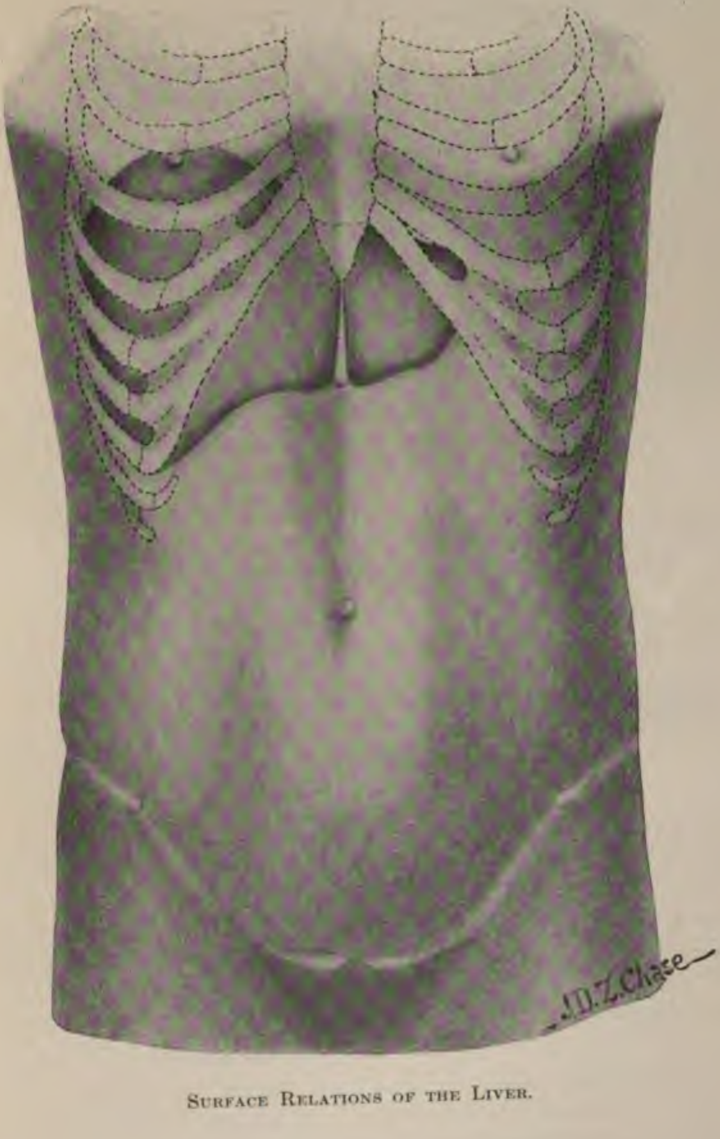
The *gall-bladder* projects from the under surface of the liver opposite the ninth right costal cartilage, close to where the inferior edge of the liver passes under the right border of the ribs, and therefore just within the right nipple-line.

EXAMINATION OF THE LIVER

INSPECTION

The body should be in the dorsal position, with the shoulders moderately elevated, and should face a good light. No evidences of the liver can be seen in the healthy adult.

PLATE X



Surface Relations of the Liver.

Bulging or fulness of the right hypochondrium, or also of the region below the border of the ribs and of the epigastrium, indicates *enlargement of the liver.* In very young persons, owing to the flexibility of the lower ribs, moderate hepatic enlargements are more noticeable, while in old persons only the most marked enlargements cause visible projection of the stiffer ribs. A more or less thin abdominal wall, moderately full in contour, also contributes to a plainer visibility of enlarged liver. The downward movement of the latter during inspiration is characteristic. In some instances the fact that the enlargement of the liver is *uniform* may be detected by the even surface of the swelling, as from *amyloid* or *fatty degeneration, leukæmic* or *malarial* hyperplasia; or that it is *irregular,* lump, or nodular, may be significant of *cancer, abscess, hydatids,* or *syphilitic* disease.

If the bulging is especially marked below the margin of the ribs, without projection of the latter, *downward displacement* of the liver is indicated. This may be caused by and associated with marked emphysema of the right lung, with pleural effusion, empyema, or pneumothorax of the right side, subdiaphragmatic abscess, or even with a concomitant heavy enlargement of the organ.

The *lower edge* of the liver may sometimes be seen as a linear shadow or sharp projection, movable with respiration, in cases of enlargement or displacement, as from "wandering liver," which occurs in certain women with general ptosis of the abdominal organs and soft, flabby relaxation of the belly walls.

A hepatic *venous pulsation* may be visible, accompanying the enlargement of passive congestion due to valvular incompetency, as tricuspid regurgitation.

PALPATION

By touch, the edge of the liver can hardly be felt in the normal individual of moderate abdominal wall thickness, either at the lower border of the ribs or where it crosses the epigastrium (dense fibromuscular resistance). In women having thin, lax abdominal walls, however, the lower border of the liver may be felt satisfactorily, especially during the downward inspiratory movement. In children, it may be felt with comparative ease.

The usefulness of palpation of the liver, over against the other methods, makes it important to practise its technic with extreme care, frequency, and patience. The patient must be in a relaxed dorsal position. The examiner's hands, indubitably warm, may first grasp the abdominal wall gently so as to estimate its thickness an resistance for a proper allowance. The finger-tips are applied flatl to the abdominal wall below the ribs, approaching upward, whil sitting at the patient's right side. A fold of skin may now be pushe up, and then immediately the fingers are depressed sufficiently to fee the lower edge of the liver, especially just after it descends with inspiration. Abrupt, poking efforts must be avoided so as to eliminat the interference of hardening of a contracted rectus muscle. Anothe way is to stand facing the feet of the patient, and hook the finger around the rib borders, and thus feel the hepatic margin pushin under their tips with inspiration.

Palpation of the liver enables one to judge of its *size* and *form* its *surface* and its *consistency*.

(1) **Size and Form.**—These are determined mainly by studyin the *lower edge* of the organ. When the long, distinct edge of th liver is felt below the border of the ribs, it may mean either *enlarg ment* or *displacement*, or both, depending upon the associated conditions which might cause one or the other, or both. If, at the sam time that the lower border of the liver is found to be depressed, percussion discovers that the upper border is not depressed, then o course the organ is enlarged. If, on the other hand, it can be demonstrated that there is a right-sided pleural effusion, pneumothora subdiaphragmatic abscess, or even marked emphysema, it is the probable that the inferior border is depressed because of displacement of the liver, although in the last-named disease there is usuall a simultaneous enlargement from passive congestion of the liver. A pointed out by Vierordt, however, "when a liver is markedly displaced downward, the impression is easily made that it is also enlarged, because by traction about its transverse axis it becomes parietal to a larger extent." Downward displacement of the lower borde of the liver may also be found as the result of tight lacing ("corse liver"), and of a general laxness of the abdominal viscera ("wandering liver"). In the latter case the liver may be pushed upward wit the finger.

An approximate idea of the *form* of the liver may be obtaine by noting the character of its edge, and the trend the latter take Much of this recognition depends upon the extent to which the live is closely apposed to the abdominal wall. The uniform and irregular enlargements have already been mentioned. A seriatim indication of the principal conditions affecting the liver edge is her given:

(*a*) Passive Engorgement (*later stages of uncompensated heart disease, and in advanced emphysema*).—The liver edge protrudes slightly, is smooth and well defined, and takes the direction of the normal virtually.

(*b*) Fatty Liver.—The organ is uniformly enlarged and normal in shape. The inferior border is smooth and slightly rounded, and is felt lower down than the congested liver.

(*c*) Hypertrophic (Biliary) Cirrhosis.—Considerable enlargement may be noted here, the lower hepatic border being often felt as low as the umbilical level, and having a smooth, well-defined, thickened outline.

(*d*) Amyloid Liver.—This is also a large liver having a smooth, often sharp lower border, or perhaps slightly rounded. The same features are usually present in

(*e*) Diffuse syphilitic hepatitis, and in

(*f*) Leukemic liver.

(*g*) Cancerous Liver.—Marked enlargement is present, and the lower border is thickened, irregular in outline and direction, nodular and notched.

(*h*) Syphilis of the Liver.—This may have some of the characteristics of the preceding condition, with perhaps less massiveness of outline, and more irregularity, especially in deep fissuring of the edge.

(*i*) Abscess may be Single or Multiple.—In cases of the latter the liver may exhibit uniform enlargement. In some instances the abscesses may be palpable as distinct prominences from the under surface of the liver, especially the right lobe.

(*j*) Hydatids.—The edge of the liver affords their discovery when sufficiently large, parietal, globular, and subject to signs of fluctuation.

(2) **Surface.**—The liver surface can be perceived in cases of sufficient enlargement, with thinness and looseness of the belly wall. When *smooth* and *even*, the liver may be engorged, fatty, leukemic, amyloid, or malarial. A *granular* surface indicates cirrhosis, best felt, of course, in the contracting stage of a hypertrophic liver before sufficient atrophy has taken place to draw it beneath the ribs. The common association of ascites with the atrophic cirrhosis makes it necessary, in order to palpate the under surface of the liver at the rib border, to displace the liquid by a brusk poking movement with the tips of the fingers.

Irregular, nodelike elevations in a thin, emaciated consumptive would hint toward *tuberculous peritonitis.*

A largely *nodular* or *bosselated* surface betokens secondary can
of the liver. The tumors vary in size from a cherry to a walnut,
even a small apple; they are rough, and not infrequently mani
to the touch small depressions on their summits (cancer nav
When a distinctly *lobulated* liver is felt, with flat elevations, a syp
litic gummatous organ is probable. Superficial abscesses and echi
coccus cysts may be inferred with round, smooth, more or less fluc
ating eminences.

(3) **Consistency.**—This is uniformly and moderately increased
chronic engorgement, leukemia, and hypertrophic cirrhosis; uniform
and markedly increased in amyloid liver; uniformly non-resistant a
somewhat elastic in fatty liver; irregularly increased in firmness
cancer and syphilis, according to the amount of scirrhous and c
nective tissue in these respective conditions. Abscess and hyda
cysts give rise to more or less doughy sense of fluctuation, and t
so-called *hydatid thrill* may sometimes be detected over large sup
ficial cysts—a peculiar whizzing sensation elicited by giving stacc
or short, quick strokes with the fingers.

The *gall-bladder* cannot be palpated normally. When distend
moderately, it may be felt as a small, soft, ill-defined, rounded m
at the edge of the ninth right costal cartilage. A distinctly palpab
enlarged gall-bladder occurs as a pear-sized and -shaped, smoo
often soft, tumor. The narrow or fixed portion of the tumor cor
sponds to the costal intersection of a line drawn from the rig
acromion process to the navel, just outside the right edge of the rig
rectus muscle. The large or dependent end is movable in the
of a circle; it may be moved almost or quite to the median line, a
up and to the right under the liver, but not downward. It is, ho
ever, movable downward with deep inspiration along with the liv
which is characteristic.

The principal causes of enlarged gall-bladder may be enumerat
as follows: (*a*) Distention by fluid from some obstructive disease
stenosis affecting the common or cystic ducts, or from inflammati
causing biliary engorgement, retention of mucus (*hydrops*), or p
(*b*) distention by gall-stones; (*c*) malignant or benign growths.

Distention of the gall-bladder by fluid accumulation feels soft a
elastic, and the tumor may sometimes be diminished by slowly
pressing part of its contents into the duodenum. Moderate enlar
ment with density of feel may indicate inflammatory thicken
The presence of gall-stones gives rise to a sensation of movable nodu
("bag of nuts") if the abdominal wall is thin and lax enough;

TABLE FOR THE DIAGNOSIS

SIZE OF THE LIVER.		CONSISTENCY OF THE LIVER.			BORDER OF THE LIVER.			SURFACE OF LIVER.	
Diminution.	Enlargement.	Soft to fluctuating.	Coarse, a little harder than normal.	Hard.	Smooth to sharp.	Thick, rounded.	Uneven lobular.	Smooth.	U ev
Simple atrophy. Atrophic nutmeg liver. Cirrhosis. Syphilis of the liver (atrophic form rather rare). Acute yellow atrophy of the liver.	Abscess of the liver. Diabetic liver. Icterus liver. Weil's disease. Fatty liver. Passive hyperemia. Syphilis of the liver. Leukemia. Connective tissue hyperplasia of the liver. Amyloid liver. Carcinoma of the liver. Echinococcus of the liver.	Fatty liver. Abscess of the liver. Echinococcus unilocularis.	Simple atrophy. Icterus liver. Hyperemia.	Cirrhosis. Syphilis of the liver. Connective tissue hyperplasia. Echinococcus multilocularis (becoming soft). Amyloid liver. Carcinoma.	Fatty liver. Icterus. Hyperplasia (sometimes slightly rounded). Echinococcus. Simple atrophy.	Fatty liver. Hyperemia. Amyloid.	Cirrhosis (only in rare cases at all palpable). Abscess. Carcinoma. Syphilis of the liver.	Hyperemia. Fatty liver. Icterus liver. Elephantiasis. Amyloid liver. Leukemia. Diabetic liver. Acute yellow atrophy of the liver.	Ci rho Ab sce Syp ili of t live Ca cino Echi cocc

NOTE.--The order of the diseases in these columns is generally so arranged that the farther down in

DISEASES OF THE LIVER (AFTER LEUBE)

Jaundice.			Ascites.		Pain.	Enlargement of the Spleen.	
Absent.	Rare.	Frequent.	Absent.	Present.	Present.	Absent.	Present.
Amyloid. Adhesive pylephlebitis. Fatty liver.	(Only if bile ducts are directly affected by the pathologic process in:) Echinococcus unilocularis. Syphilis of the liver.	Abscess. Hyperemia. Cirrhosis. Carcinoma. Echinococcus multilocularis. Elephantiasis hep. Icterus liver.	Fatty liver. Elephantiasis hep. Icterus liver. Echinococcus unilocularis. Abscess.	Carcinoma. Syphilis with cicatrices. Echinococcus multilocularis. Cirrhosis. Pylephleb. spec. adhesiva. — Amyloid (through the original process). Hyperemia (in later stages constant).	Echinococcus multilocularis. Acute yellow atrophy of the liver. Carcinoma. Syphilis of the liver. Abscess.	Carcinoma. Fatty liver.	Echinococcus unilocularis (rarely by stasis in the portal vein system). Hyperemia of the liver. Syphilis of the liver. Cirrhosis. Echinococcus multilocularis. Hypertrophic cirrhosis. Amyloid liver. — Furthermore, through general infection caused by acute yellow atrophy of the liver. Abscess of the liver. Weil's disease.

respective symptom is the more characteristic of the special affection of the liver,
column it is quoted.

distinct friction or grating sensation may be palpable. A firm, irregular mass signifies carcinomatous thickening or growth.

The fluid distention of the gall-bladder with bile may be the result of gall-stones in the common duct, or of an obstructive duodenal catarrh, or of a roundworm (*ascaris lumbricoides*), or of some growth adjacent compressing the duct, as cancer of the head of the pancreas.

Differential Diagnosis.—While tumors of the gall-bladder may be simulated by hydatid cysts, and by abnormal prolongations of the lower border of the liver, these conditions occur so rarely as to need no more than the mentioning here.

Movable or floating kidney, which is more often found on the right side, has frequent and important differential bearing. Both it and the distended gall-bladder are smooth and rounded, but the former is characteristically bean-shaped, the latter pear- or gourd-shaped. The kidney has also the notch of the hilum, and if much displaced, in some instances the pulsations of the renal artery may be felt. The kidney has a uniformly fleshy feel, whereas the gall-bladder may be soft with liquid, rough with calculi, or hard and nodular with cancer. Again, the pear-shaped gall-bladder is usually so superficial that it may be felt by direct palpation in front, while the displaced kidney is more deeply palpable, and usually requires counter pressure through the soft lumbar tissues from behind. Also, the kidney has a wider range of mobility, particularly in a vertical direction, whereas the enlarged gall-bladder swings in a short, circumscribed arc, with its long axis as the radius; it is frequently possible to slip the kidney under the fingers in various directions. Respiratory mobility affects the distended gall-bladder more than it does the kidney unless, of course, the latter be floating far below the liver. Finally, gaseous distention of the colon makes the gall-bladder prominent, the kidney less so, or completely prevents it being felt.

PERCUSSION

There are two so-called *areas of liver dulness*, one, the area of *absolute dulness*, representing that portion of the organ which is parietal—that is, in apposition with the lower ribs without the intervention of lung tissue; the other, the area of *relative dulness*, representing the narrow zone above and adjoining the absolute area, where the liver is covered by a thin layer of lung. The vertical extent of absolute liver dulness is widest in the nipple-line, beyond which it tapers gradually around the chest to an interspace in width the right scapular line, and more sharply to the left in fron until it passes into the cardiac dulness, from which it cannot definitely delimited, at about the sixth rib within the left par sternal line.

The *lower border* of normal liver dulness corresponds closely wi its anatomic limits; that is, from the right midclavicular line bac ward it begins at the borders of the ribs (tenth rib in the axilla line, eleventh rib in the scapular line). Here, anteriorly, it is nec sary to percuss gently from below, so as to avoid eclipsing the fir signs of dulness of the thin edge of the liver by the adjacent colon tympany.

The *upper border* of absolute liver dulness—that is, the lung-liv boundary—naturally begins where lung resonance absolutely cease at the base of the ensiform cartilage in the middle line, at the six rib in the nipple-line, the eighth rib in the midaxillary line, the ten rib in the scapular angle line. This is at the same time the *low border* of *relative dulness*.

The *upper border* of *relative dulness* is about one rib higher around; and above the fifth, seventh, and ninth ribs, in the respecti lines just mentioned, passes at once into clear pulmonary resonanc

Posteriorly, near the spine, the lower border of liver dulness ca not be separated from the kidney dulness. In the epigastric regi it is often difficult to ascertain the lower border of liver dulne because of confusing gastric and intestinal tympany, on the one han and because of dulness of firm, thick bellies of the rectus muscl on the other.

We percuss downward from the lung to determine the upper bo der of hepatic dulness, and upward from the abdomen to determi the lower border. The normal boundaries vary somewhat in differe individuals, according to the size and shape of the liver, and t condition of the lungs and adjacent organs, as well as the formati of the bony chest case. Besides, the upper border of dulness displaced by deep breathing (active mobility).

The total width of liver dulness (absolute and relative combine is about four inches in the left midclavicular line, six inches in t midaxillary line, and three inches in the scapular line. In childre the lower border of dulness is normally lower than in adults, and t upper border higher, because of the relatively greater size of the orga *Per contra*, the lower limit of hepatic dulness is a little higher the aged, because of senile shrinkage.

Auscultatory percussion may be applied with precise results in outlining the liver borders, and in determining whether an adjacent tumor is an outgrowth of the liver itself, or whether it is conjoined to a neighboring viscus.

Pathologic Relations of Changes of the Area of Liver Dulness.—(1) The UPPER BOUNDARY may be found to be *higher* than normal. In the first place, this may be due to some thoracic condition, and thus becomes an *apparent* instead of an actual increase of liver dulness upward. The causes may be lobar pneumonia, pleural effusion, tumors of the lung and pleura, marked thickening of the pleura, subphrenic abscess, and retraction of the lung, causing more of the liver to become parietal. In cases of large pleural effusion, the liver is displaced downward, with consequent lowered inferior boundary of dulness, and extensive total dulness from the top of the effusion to the lowest point of liver dulness. With small, beginning pleural effusions it may be difficult to eliminate liver dulness extended upward unless the history and other evidences point to thoracic disease.

Real displacement upward of the upper boundary of liver dulness may occur from marked *enlargement* of the organ, *tumor* of its convexity, or *upward displacement.* In the former state the lower boundary is simultaneously deeper than normal. A tumor is indicated if the raised upper boundary of dulness is irregular in outline, or a large abscess or hydatid cyst may be present. The lower border of liver dulness may also be depressed moderately. Displacement of the liver upward, as from ascites or intense tympanites, is accompanied with upward displacement of the lower border of liver dulness, and less area of dulness generally, because of the diminished extent of the organ, which is parietal.

(2) UPPER BOUNDARY of liver dulness is *lower* than normal. This results from any condition in which there is encroachment downward of the extent of clear sound over the right thorax. If at the same time the lower boundary of liver dulness is displaced downward, the cause is either marked *emphysema* or *pneumothorax.* In the former condition the simultaneous low position of both boundaries may be accompanied with decided low position of the lower border also, because of the enlargement of the liver due to the engorgement which usually goes with advanced emphysema.

A low position of the upper boundary of liver dulness without simultaneous lowered inferior border may be found temporarily as an accompaniment of *compensatory emphysema*, where the lung fills the complementary pleural sinus above the diaphragm.

(3) The LOWER BOUNDARY of liver dulness is *higher* in upward displacement of the organ, as from ascites, tympanites, and enormous abdominal tumors pressing upward; also, in atrophy (cirrhotic) of the liver.

(4) The LOWER BOUNDARY of liver dulness is *depressed* in enlargement and downward dislocation, as has already been pointed out in connection with associated displacement of the upper boundary. The lower line of dulness is more vertical or horizontal according to the cause, degree, and side affected in depressing the organ. A large pleural effusion on the right side will produce the greatest tilting downward of the right lobe of the liver, and consequently a sharp slope upward toward the left of the lower border of dulness. On the other hand, a heavy and abundant pericardial effusion will cause the lower border of liver dulness to take a more horizontal trend because of the pressure upon the left lobe.

DIMINISHED TOTAL AREA OF LIVER DULNESS is most commonly caused by atrophic *cirrhosis* of the organ; rarely by *acute yellow atrophy*. An apparent decrease in the area of hepatic flatness may be due to encroachment by distended (emphysematous) lung above, or by distended stomach or colon, or pneumoperitoneum (from ulcerous or traumatic perforation of the gut) from below.

Absence of liver dulness indicates a complete dislocation of the liver, either as wandering liver or as a part of inversion of the viscera.

Apparent enlargement of the liver downward may be misjudged because of contiguous dull areas, such as arise from fecal accumulations in the transverse colon, tumors of the colon or stomach, renal and large ovarian tumors, pancreatic cyst, tumors of the omentum (sarcomatous or tuberculous), and ascites. It may also be mistakenly inferred on account of a moderately prolapsed position of the organ.

Two alterations of the *relative liver dulness*, of minor diagnostic value, may be noted: it is *high* in association with great enlargement upward of the liver, and consequent high vertical position of the anterior wall of the diaphragm; it is *low* from pneumothorax and severe emphysema, where the diaphragm is thus flattened down so that its anterior portion leaves the thoracic wall somewhat perpendicularly (Vierordt).

AUSCULTATION

By this method one may discover a *perihepatitis* by recognizing fine *crepitations*, even *rubbing* or *crumpling* sounds in the lower interspaces, between the right parasternal and axillary lines.

A continuous, soft blowing *murmur* may be heard over the liver in some instances of tricuspid regurgitation.

A *gall-stone crepitus* or grating sound may be audible over the gall-bladder in certain cases during deep breathing, palpation, or movable pressure with the stethoscope.

TOPOGRAPHIC ANATOMY OF THE SPLEEN

The spleen has an oval shape, its long axis being parallel with the ribs under which it lies in the left infra-axillary region, these ribs being the ninth, tenth, and eleventh. Posteriorly, it reaches nearly to the spine, about one inch from the tenth dorsal vertebra. Laterally, it approaches closely to the midaxillary line, so that its length averages about four and one-half inches, with its corresponding axis running a course downward and forward. The spleen's breadth

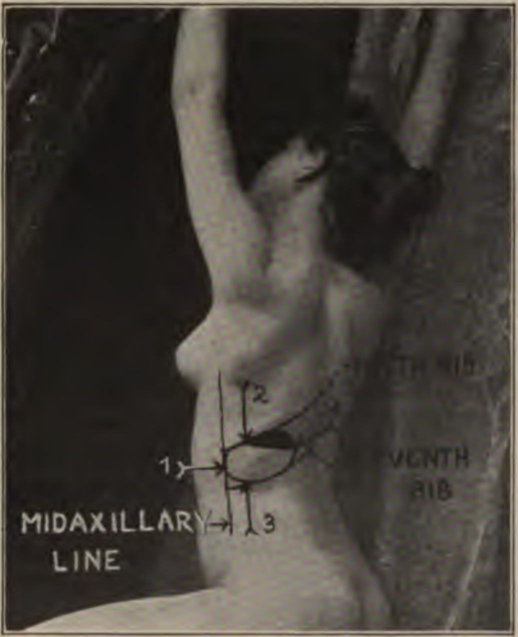


FIG. 83.—SHOWING THE SURFACE TOPOGRAPHY OF THE SPLEEN. Shaded area = portion overlapped by lung. Numbered arrows show the lines along which percussion should be conducted. (Butler.)

is about three inches. The notch on its anterior border is palpable only when the organ is enlarged.

Its *relations* to other structures should be recalled. It lies in close contact with the under surface of the diaphragm as it arches upward. Postero-inferiorly, the spleen overlaps a small portion of the left kidney, and anteriorly, it lies in contact with the outer portion of the greater curvature of the stomach and the splenic flexure of the colon. Topographically, the spleen is in contact with the lower portion of the thorax only with its lower two-thirds, the upper third being separated from the ribs by the diaphragm and lower border of the left lung. This parietal portion is somewhat variable on account of the mobility of the lung as affected by the depth of the respirations and the posture of the body. The upper and anterior border of the spleen forms the *spleen-lung angle* at about the ninth rib in the posterior axillary line, when the body is in the dorsal position, or it may be at the anterior axillary line when in the right-sided position. The lower splenic border runs backward along the eleventh rib.

EXAMINATION OF THE SPLEEN

INSPECTION

From its position beneath the ribs, it is obvious that the *normal spleen* can neither be inspected nor palpated. Only when greatly enlarged can the spleen be inspected, because of the bulging downward and forward from under the left hypochondrium, often distending the greater portion of the abdomen. The antero-inferior edge of such a greatly enlarged spleen may exhibit a sharply outlined, oval-shaped border of slight elevation of the overlying skin. This edge and the whole tumor move with respiration. I have noticed even a slight depression, corresponding to the splenic notch, in two cases of enormous splenic enlargement where the belly was tensely filled.

PALPATION

The ease and accuracy with which the pathologic spleen may be palpated depends upon the degree of its enlargement, the technical skill of the examiner, and the resistance of the abdominal wall. Slight and moderate splenic enlargements call for a practised and patient technic for their detection. Under these circumstances the antero-inferior edge may be felt to project just beyond the free border of the ribs, especially during a deep inspiration, as a smooth, rounded, blunt, rather firm but not hard surface.

The patient should be in either the recumbent or the midway position between the dorsal and right-sided position. The warm hands may be applied in several ways: Ordinarily one may apply the fingers of both hands close together under the left rib border, in the midaxillary line; thus moderate enlargements may be detected, such as the *acute splenic tumor* of typhoid fever. It is advisable in this method to draw the abdominal skin downward a little first, so as to palpate through as lax tissue as possible, and to feel for the edge of the spleen, especially at the moment of fullest inspiration.

Probably the most satisfactory and reliable method, however, is to palpate bimanually by crossing hands, so that the left, placed over the normal position of the spleen, draws this lower portion of the thorax inward and downward, at the same time sliding the skin loosely toward the right, for the reason given in describing the preceding method. The right hand of the examiner meanwhile awaits the spleen by pointing straight up under the rib border, and pressing in a little with the finger-tips at the end of inspiration, while the pressure of the left hand over the splenic region is exerted simultaneously.

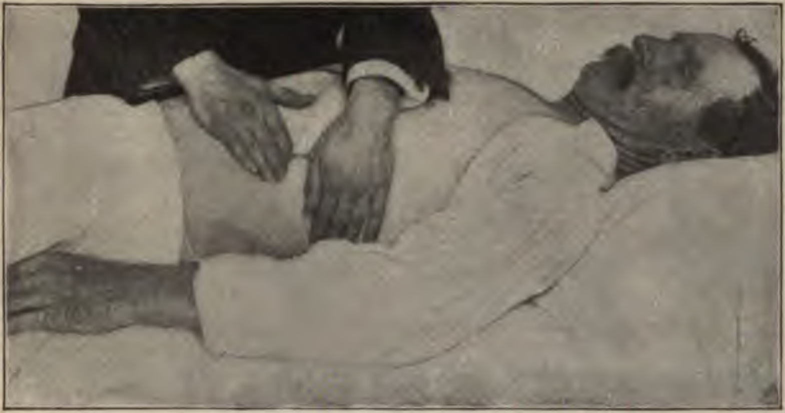


FIG. 84.—PALPATING THE SPLEEN. (Musser.)

Another method is to have the patient lie on the right side, while the physician stands behind and crooks the fingers under the rib margin and tries to feel the spleen at the end of a deep inspiration.

Abnormally, the spleen can be felt:

(1) If it is enlarged.

(2) If it is displaced (wandering or floating spleen).

(3) In certain cases of chest deformity.

By palpation we determine the *size, shape, surface, consistence, mobility,* and *position* of the spleen. As ordinarily the spleen is palpable only when it is enlarged, it will be appropriate to point out the various causes of enlargement, and at the same time indicate the changes in the other physical signs associated with a given variety of enlargement. Most of the enlargements, especially those of slight or moderate degree, are uniform in shape; and even the majority of the larger and more chronic enlargements have the form of the normal spleen.

(*a*) *Infectious Febrile and Septic Diseases.*—These cause *slight or moderate* splenic enlargement, of uniform shape, smooth surface, soft or moderately firm consistence, and slight respiratory mobility. The principal causes under this head are typhoid fever, malarial fever, scarlet fever, smallpox, relapsing fever, typhus fever, erysipelas, septicopyemia, acute miliary tuberculosis, and epidemic cerebrospinal meningitis.

(*b*) *Chronic Blood Diseases, Principally.*—Here there is also uniform enlargement of the spleen, but the size is greatly increased, the surface likewise smooth or but slightly uneven, the consistence hard, and the mobility easily demonstrable unless the enlargement is so excessive as to prevent downward movement of the diaphragm. To be enumerated here are the following: Chronic malaria, chronic congestion from primary hepatic cirrhosis, leukemia, splenic anemia, Hodgkin's disease, amyloid disease, tuberculosis, syphilis, rickets.

(*c*) *Neoplasms, etc.*—The spleen is irregular in shape, moderate or marked in enlargement, uneven of surface, firm, fluctuating, or boggy in consistence, and more or less responsive to respiratory movements. Thus we have cancer, abscess, and hydatids of the spleen.

In most of the cases of large, uniform enlargements of the spleen, notches may be felt along its anterior border.

Wandering spleen may be palpated better sometimes by placing the patient in the knee-chest position; the hilum may also be felt, and respiratory mobility is diminished, or more often absent.

Differential Diagnosis.—It is important to distinguish between splenic enlargement and tumor of the left kidney. This will be discussed in the consideration of the latter organ.

It is possible sometimes to mistake at first a *fecal mass* in the *splenic flexure* of the colon for an enlarged spleen; but the peculiar doughy feel of the former, and its removal by catharsis or enemata, as well as the absence of splenic outlines and of respiratory and passive mobility, serve to make the diagnosis clear.

The occurrence of cancer of the cardiac end of the stomach, or of an enlarged left lobe of the liver, should be borne in mind, but hardly needs any specific points of differentiation from splenic tumor at this place other than to mention that the study of location, form, consistence, and mobility will usually suffice to decide in each instance, associated, of course, with the gastric history and symptomatology, and the results of other methods of examination.

PERCUSSION

This method of examining the spleen is of less value than palpation, and is only confirmatory of enlarged spleen when this organ has been felt as such.

The *normal splenic dulness* represents that portion of the spleen which is parietal; that is, below and between the inferior lung border and the eleventh rib. This dulness is not extreme or flat in quality, owing to the juxtaposition of the resonant lung and the tympanitic stomach and colon. The dull area varies slightly in position and definiteness because of its respiratory and passive mobility, being more posterior when the patient lies upon the back, more anterior but less dull when on the right side (the spleen being farther from the ribs); with the patient standing, however, the spleen is a trifle lower.

To *outline* the *splenic area of dulness* by percussion, the subject should lie on the right side, midway between the dorsal and extreme lateral positions, with the left arm elevated. As the dulness obtained is merely relative in most cases, the percussion should be made with light strokes over the antero-inferior portion (uncovered by lung); posteriorly, stronger strokes are necessary. When the spleen is greatly enlarged, the dorsal posture will do.

To determine the *upper border* of splenic dulness, we begin by noting the inferior lung limit. With moderately strong blows we percuss sufficiently high up in the posterior part of the axillary region to elicit good pulmonary resonance. Percussing thence downward, we note the first evidence of slight elevation of pitch and relative dulness, then with lighter strokes continue downward, noting true dulness, until a faint tympanitic quality is perceived. This gives the upper boundary of splenic dulness, where relative passes into nearly absolute dulness, and also the vertical extent of dulness along the perpendicular line of percussion.

The *anterior border* of splenic dulness is obtained by percussing from before backward at the level of and in the direction of the tenth rib, beginning at the costal margin. Thus we note where the gastric tympany changes to splenic dulness.

The *inferior limit* of splenic dulness is determined by percussing upward in about the posterior axillary line until intestinal tympany merges into the dulness, which is normally at the eleventh rib.

The *posterior limit* seldom needs to be determined, and usually cannot be satisfactorily, for obvious anatomic reasons.

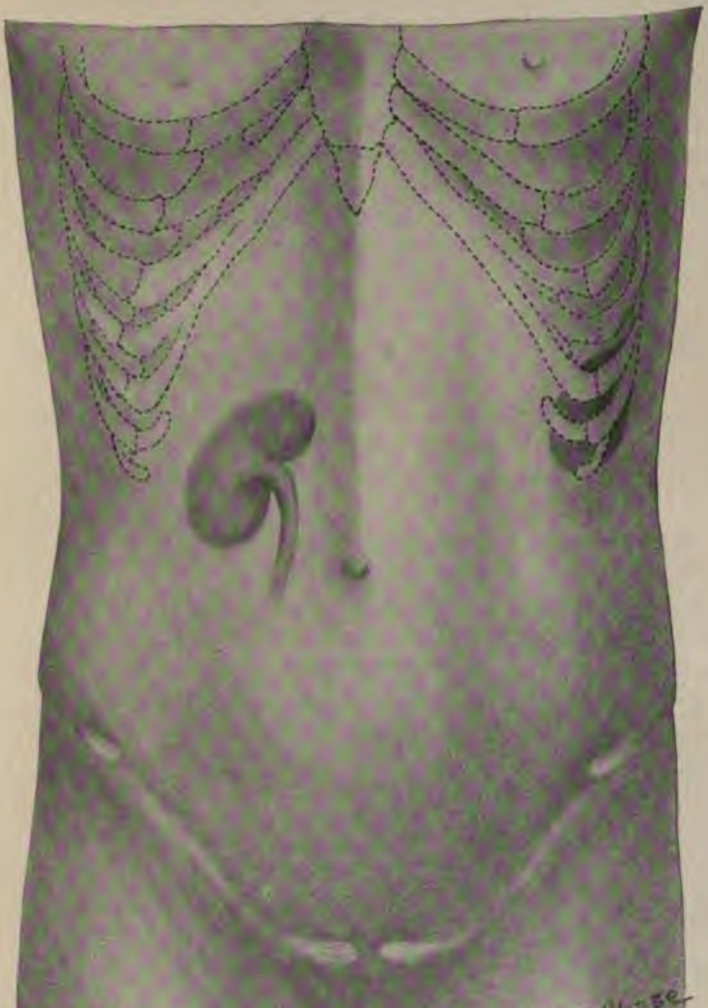
The *size* of the area of splenic dulness is estimated ordinarily by its vertical extent in the middle or posterior axillary line, below the spleen-lung angle, where the inferior lung border begins to overlap the spleen. Normally, this measures on the average about three or three and one-half inches. Again, the anterior limit of the splenic area should not extend beyond the *linea costo-articularis*, or the line drawn from the tip of the eleventh rib to the left sternoclavicular articulation (Vierordt).

Variations.—The area of splenic dulness may *apparently* be increased through distention of the stomach with food, fecal masses in the adjacent colon, colloid cancer of the cardia of the stomach, left pleural effusion, pneumonic consolidation at the left base, pleural thickening, or a new growth of the pleura or lung.

On the other hand, splenic dulness is delimited with difficulty in obese persons, and it is *apparently decreased* in size by encroaching emphysematous lung, by pneumothorax, tympanites, and by displacement upward by ascites or a large abdominal tumor. *Total absence* of splenic dulness may be due to *floating spleen*, which may then be palpated in its abnormal position, and recognized by its shape, mobility, notches, sharp edge, and the reappearance of dulness in the proper area when the organ is replaced.

Enlargement of the Splenic Dulness.—After eliminating the possible sources of error mentioned above in connection with an apparent increase of dulness, the examiner should note the shape of the increased area of dulness, and its position relative to the adjacent organs and ribs. Enlargement of the spleen may be diagnosed if the vertical measurement of dulness is four inches or over, and if the anterior border of the dull area extends beyond the linea costo-articularis. In instances of great enlargement the gastric tympany may be entirely obliterated.

PLATE XI



SURFACE PROJECTION OF THE RIGHT KIDNEY: ANTERIOR RELATIONS OF SPLEEN.

AUSCULTATION

A *friction sound* over the splenic area may be set up by a plastic deposit upon the serous covering of the spleen and the parietal portion of the peritoneum opposite to it, this sound corresponding to the diaphragmatic movements. Such evidence of *perisplenitis* may indicate a septic embolus and infarct of the spleen, an abscess, or simply an intense splenic congestion associated with a severe infectious fever. In the marked cases of enlargement, as in leukemia and chronic malaria ("ague-cake"), distinct perisplenic friction may be heard not infrequently with the stethoscope.

TOPOGRAPHIC ANATOMY OF THE KIDNEYS

These bean-shaped organs lie against the posterior abdominal wall, one on each side of the spinal column. They are embedded in fat as they lie in their respective retroperitoneal fossæ, upon the quadratus lumborum muscles and the lumbar portions of the diaphragm, the right, however, a little lower than the left—about ½ in. Their lower ends diverge somewhat, so that they are about a finger's breadth farther from the median body line than their upper ends. Each kidney is about 4 in. long, about 2¼ in. broad, and ¾ to 1 in. thick. The upper border of the right kidney lies in contact with the liver, and reaches the level of the eleventh dorsal spine; that of the left kidney, overlapped by the spleen, reaches to the eleventh rib. The lower end of the left kidney coincides with the level of the third lumbar spine; that of the right is ½ in. lower, or about

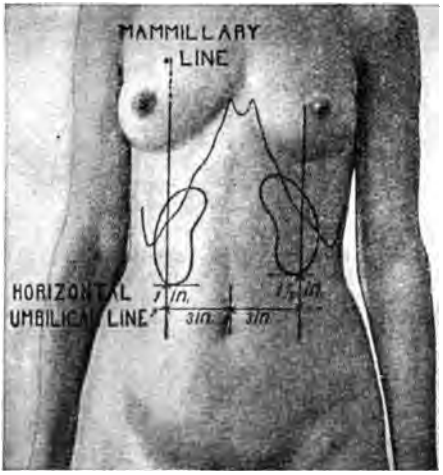


Fig. 85.—Showing the Normal Surface Relations of the Kidneys Anteriorly, and the Method of Determining these Relations. (Butler.)

Slight or *moderate enlargements* are not palpable unless the ki is at the same time displaced. These include such affections as engorgement, chronic nephritis ("large white kidney"), and loid kidney.

Large renal tumors are difficult to diagnose, because of their trusion forward and simulation of other abdominal organs adja The following enlargements of the kidney are palpable withou dislocation: Malignant disease, sarcoma more often than carcin cystic degeneration; hydronephrosis; pyonephrosis, including tub losis; echinococcus disease, and perinephric abscess.

These tumors are felt to occupy the ordinarily soft hollow o loin, sometimes extending anteriorly nearly as far as the umbil and filling completely the space between the costal margin an crest of the ilium, as in a case of sarcoma which I saw in a of eleven years, and one of cystic disease of the left kidney in a y woman. Sarcomata in children are especially large tumors as a ing the kidney.

The new growths are usually firm in consistence, and give kidney an irregular shape and uneven surface. With hydro- pyonephrosis, however, the outline is round and smooth, and fluctuation may be elicited; likewise with hydatids, if the cysts sufficiently large. Renal tumors are but slightly, if at all, influe by the respiratory movements, and the absence of descent with spiration in the case of a large tumor in either flank points to kidney rather than to splenic or hepatic involvement. With the nephric abscess there is a boggy resistance over the region of kidney. In tumors of moderate size, as in certain cases of c disease, the shape of the kidney may be preserved to a degree, rounded edge, and a sort of *ballotement* phenomenon may be dete by bimanual palpation. An important aid in the diagnosis of r tumors is the relation of the colon, and often the small intes which may be felt and percussed, thus showing that they are in fr while, on the other hand, splenic tumors especially, and hepatic mors usually, do not have bowel between them and the abdominal In cases of doubt, the colon may be inflated with air per rectal with bulb attachment.

PERCUSSION

Although a less useful source of information as to the phy status of the kidney than palpation, nevertheless percussion is often neglected when additional and confirmatory results may when it is at once enlarged and displaced. It is then usually seen in either flank, in the waist line, or even lower down. The most common causes of renal tumor with bulging in the anterolateral lumbar region are sarcomas in children, and *cystic* or *hydronephrotic growths* in adults. They are usually not movable with respiration. Sometimes a *perinephric abscess* may be inferred by the discovery of a roundish, shiny, edematous-looking swelling in either renal region.

PALPATION

This is certainly the most informing method of examining the kidneys physically. Normally, it is possible to palpate them bimanually in some children, and in those with thin, lax abdominal walls, especially during inspiration.

Technic.—One way to palpate the kidneys is to have the patient in the dorsal position, with slight flexure of the whole frame, from head to heels. Then, with one hand pressing firmly forward into the loin, and with the other pressing deeply (finger-tips, palmar surface) in front, near the midclavicular line, one may feel the *enlarged* or *displaced* kidney slip between the fingers as a "greasy mass," to and fro, during inspiration and expiration. This mode of palpation is usually most satisfactory in the transverse line, which connects the inner portions of the waist curves. A movable kidney may thus be grasped for one-half or more of its length, and popped back during expiration. In extreme cases the kidney may be palpated throughout its whole length ("floating kidney").

Another favorite method with many is to attempt to grasp the kidney while the patient stands and leans slightly forward, with the hands resting upon a chair or table; in this position the abdominal walls are relaxed, and the kidneys fall forward. In either the reclining or standing posture one may also palpate the movable kidney at times with one hand, by grasping the soft part of the flank, the thumb pressing under the costal margin while the fingers press forward from behind. With a deep inspiration a movable kidney may be felt to slip downward within the grip, squeezed gently (often causing a sickening sensation), and then slip upward with expiration, or be pushed up under the seizure.

Besides *movable kidney*—which, of course, may have its normal shape, more readily palpable because there is usually a deficiency of surrounding fat in such cases—there are the various *enlargements* of the organ.

wall, advancing, as they increase in size, obliquely toward or beyond the umbilicus; they also present usually a smoother, harder surface, and a sharp anterior edge with the characteristic notches. Renal tumors do not maintain the shape of the kidney; are, in fact, less likely to be uniform enlargements of this organ; they fill the flank and curve of the back more especially, and the inflated colon is demonstrably (percussion) in front of them.

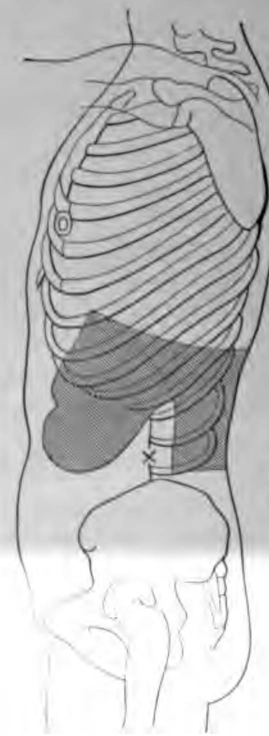


FIG. 87.—TUMOR OF SPLEEN. × Resonance between the posterior margin and the lumbar muscles. (Le Fevre.)

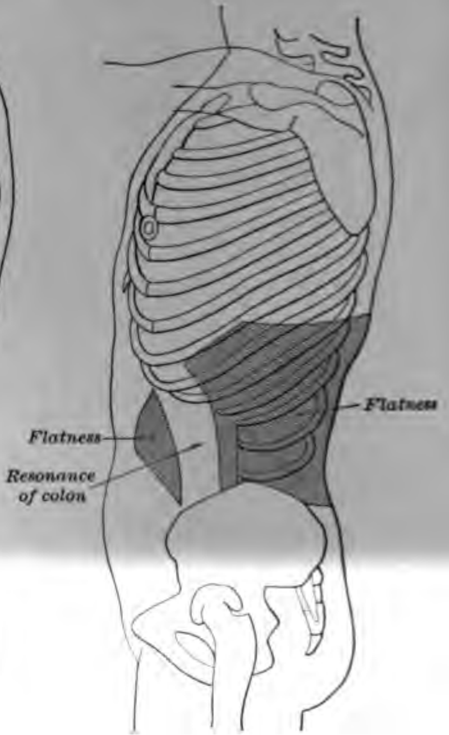


FIG. 88.—TUMOR OF THE LEFT KIDNEY. (Le Fevre.)

Moderate enlargements of the *spleen* and ***wandering spleen*** may be differentiated from *floating kidney* by the following points: In the first place, the left kidney is seldom found displaced, and hence a tumor on the left side is presumptive evidence in favor of splenic tumor. Secondly, the palpable differences of form already noted, the hilum of the kidney and the notches of the spleen. Thirdly, respiratory mobility favors the spleen. Fourthly, the percutory relations of the colon, and the results of percussion over the splenic and renal areas. If the tumor is replaceable, it speaks rather of movable kidney. Finally, a visible or palpable depression and yielding in the region of the eleventh and twelfth ribs may indicate displaced kidney.

Associated with wandering kidney frequently, and thus having diagnostic clue, are the physical signs of general displacement of the abdominal organs—*splanchnoptosis,* or Glenard's disease; sometimes, also, of dilation of the stomach, prolapse of the generative organs, and various "skeletal stigmas," such as a tendency to or the actual presence of spinal curvatures, hernias, dislocations of joints, pedal, and thoracic deformities, etc.

Obviously, in connection with all renal physical examinations much definitive aid and confirmation are obtained by the results of clinical chemistry and microscopy.

PART III

THE RÖNTGEN RAY IN MEDICAL DIAGNOSIS

CHAPTER XIX

THE RÖNTGEN RAY IN MEDICAL DIAGNOSIS

By G. E. PFAHLER, M.D.

THE increased field of usefulness of the Röntgen ray, and its increased accuracy in medical diagnosis render a work upon Physical Diagnosis incomplete without at least a chapter upon the subject.

Two objects have been kept in mind in the preparation of this chapter. First, we desire to acquaint the general practitioner and the student with what may be expected from the X-ray as an assistance in making a diagnosis. Second, the general technic and directions for making the examinations are given to assist those who are already partially familiar with Röntgen work. A complete description of the technic of Röntgenology would require too much space, and would be out of place in a work of this kind. A special effort has also been made to give directions that will assist the physician in the interpretation of Röntgen negatives.

The subjects treated will be those belonging purely to internal medicine, and will consist of a study of the thoracic and abdominal viscera.

THE RELATIVE VALUE OF RÖNTGENOSCOPY AND RÖNTGENOGRAPHY

The fluoroscopic screen renders valuable assistance in diagnosis. It can be used by those who are less familiar with the general technic of Röntgen work. It is less expensive than the making of a Röntgenogram. It enables the physician to study the movements of the organs. Finally, it enables him to study the organs from various positions, and to get his results quickly. In order to make Röntgenoscopic examinations successfully, it is necessary to make them in a darkened room, and the physician should spend from five to ten minutes in the dark room before beginning the examination, so that his eyes will be in condition to appreciate delicate shadows.

Röntgenoscopy, however, has several disadvantages. It requires an exposure of several minutes, and at times a half-hour, until the physician satisfies himself and those around him concerning the various shadows. This at once makes the Röntgen rays a serious danger to the patient, and a very grave danger to the operator, because of the many repetitions of these exposures in the examination of many patients. The danger to the operator is a troublesome dermatitis, the loss of hair and nails, and, as has been more recently shown, the danger of the production of sexual sterility. This has led the older and more experienced Röntgenologists to abandon, or at least limit, Röntgenoscopic examinations. These prolonged examinations are also a great wear upon the tubes and the apparatus, and therefore more or less expensive.

Röntgenography has many advantages. With good technic and a good apparatus, a Röntgenogram can be made of any part of the body in from one second to one minute. It is practically impossible to do the patient harm in this short time. During this examination the operator can work behind screens. We therefore avoid at once the most serious objection found in screen examinations. The plate forms a most accurate and permanent record, which can be demonstrated to others, or can be referred to at any time in the study of the progress or cure of the disease. Organs can be studied from various positions by making several plates. The records will then be permanent, and can be used for more detailed study. The most important advantage, however, of the Röntgenogram is the great improvement in detail and accuracy. Shadows which cannot be seen at all upon the screen will appear clearly upon the plate. This increase in detail and accuracy will easily repay the extra cost of time and money, and is to be recommended always, even when a fluoroscopic examination has been made.

GENERAL TECHNIC OF RÖNTGENOGRAPHY OF THE CHEST

As a rule, Röntgenograms of the chest are best made with the patient in the recumbent position. The supine or the prone position may be used. If the lesion to be especially studied is nearer the posterior wall, the supine position should be used. If the lesion is nearer the anterior wall, the prone position is to be preferred. If time and expense are not considerations, both positions should be used in each case. The hands should be clasped over the head. This throws the scapula out of the way, and leaves the lateral wall of the chest clear. When fluids within the chest are to be examined, the patient should be placed in the upright position.

The plate should be placed beneath or posterior to the patient, and the tube should be placed at a distance of from 16 to 20 in. (40 to 50 cm.). Ordinarily, the tube should be placed in the median line, on a level with the nipples. A 14 × 17-in. plate will usually record the whole chest cavity and the shoulders.

The time of exposure will vary with the organ under examination. If the lungs are being studied, the exposure should be from five to twenty seconds, or during the time that the patient can hold the breath. There are few patients needing an X-ray examination who can hold their breath longer than twenty seconds, and few who cannot hold it five seconds. A few Röntgenograms have been made in a second or less, but this cannot be depended upon as a working basis.

The ability of the patient to hold the breath should be carefully tested beforehand. The time will be found to increase after a few efforts. This test will enable the operator to estimate the time of the exposure, and will give the patient confidence.

Röntgenograms of the chest made in the fraction of a second require the most favorable circumstances, and even then the shadows are weak and indistinct. Therefore, the heart cannot be photographed, as a rule, in the resting position. Usually all of the organs of the chest can be photographed in from five to twenty seconds. If the internal structure of an aneurism or of the vertebræ is to be studied, the exposure may have to be prolonged.

The vacuum of the tube should be medium. That is, it should equal the resistance of a parallel spark-gap of from 2½ to 3 in. (6 to 8 cm.) in the open air.

The amount of current should be as great as can be forced through the tube.

Smaller sections of the chest may be examined and greater detail obtained by making use of the diaphragm technic.

Rontgenoscopy of the Chest.—If a Röntgenoscopic examination be made, the detail can also be improved by making use of a diaphragm, which will exclude all of the unnecessary rays. A protective fluoroscope, and protecting gloves and apron, should be used by the operator.

Appearances of the Normal Thorax (Plate XII, A).—Whether the physician uses the Röntgen ray as a part of his daily work, or whether he only uses it occasionally, or whether he depends entirely upon some one else for his work, it is necessary that he should become familiar with the normal appearances of the various parts of the body. Until he has done this he cannot appreciate the pathologic lesions, even when they are pointed out to him.

If a screen examination is being made, the most striking shadow will be that of the diaphragm rising and falling with each respiration. The average normal excursion of the diaphragm has been found by Williams ("The Röntgen Rays in Medicine and Surgery," 1903) to be 6.8 cm. on the right side and 7.1 cm. on the left side, between forced inspiration and forced expiration. The average normal excursion during quiet breathing, however, was found to be about 1.5 cm. In the Röntgenograph, the shadow of the diaphragm will be found resting on a level with the tenth rib, or between the ninth and tenth, in the midscapular line, providing the patient holds his breath during the rest between inspiration and expiration. This is the best position in which to hold the breath for radiographic purposes.

The next striking shadow will be that of the pulsating heart, which is a triangular shadow in the lower middle portion of the chest, and extending toward the left. In the Röntgenogram, this shadow is found to extend about 4.5 cm. to the right of the median line, and about 11 cm. to the left of the median line. This will vary somewhat with the size of the patient, and therefore with the normal size of the heart.

Having observed the shadows of the spinal column, the thoracic and shoulder bones, we are then prepared to study the more delicate shadows of the structure of the lungs. When making observations with the fluoroscope, we are struck by the marked transparency, and both sides should be equally clear. In the Röntgenograph, however, we may observe some of the structure of the lung. We usually detect shadows which are more dense in the region of the large pulmonary vessels. In exceptionally clear negatives we may see branches and subbranches (Plate XII, B). The transparency about the apices will be somewhat modified by the muscles of the shoulder, and this modification will vary with the amount of muscle. In general, the clearness of the image will be diminished in proportion to the amount of fat, muscle, or edema of the chest walls.

In the upper portion of the mediastinum the shadow of the arch of the aorta, and less clearly those of the innominate and carotid arteries, may be seen.

TECHNIC OF RÖNTGENOGRAPHY IN PATHOLOGIC CONDITIONS OF THE CHEST

THE LUNGS

Pulmonary Tuberculosis.—One of the most important applications of the Röntgen rays in internal medicine is that of aiding in the diagnosis of pulmonary tuberculosis. No degree of skill in the application of physical diagnosis can compete with the rays in determining and recording with accuracy the plane location of various pathologic lesions. Yet each has a distinct value of its own, which cannot be replaced. Each case, if possible, should be carefully studied by both physical and Röntgen examination. As a rule, a careful physical examination should precede the Röntgen examination, not because it will modify the shadows, but because it will enable the physician to interpret those present, may modify the method of examination, and thus make a second examination unnecessary.

Cases in Which This Examination Is Indicated.—No case is thoroughly studied without this examination. It is particularly indicated in early or doubtful cases. It should be our aim to make the diagnosis in the prebacillary stage (before bacilli appear in the sputum), or even in the presputal stage (H. S. A.), and here the rays will be of the greatest assistance. In favorable cases the earliest possible lesions can be shown; that is, during the stage of congestion (Plate XII, B). It is also important to use the rays to make an accurate record, and estimate the progress or cure of the disease. Old calcified tubercles, old scars, consolidations, abscesses, cavities, emphysema, gangrene, thickened pleura, pleural effusion, pneumothorax, and enlarged mediastinal glands may be observed.

Interpretation.—The proper interpretation of a negative is as important and as difficult as the making of it. The negative itself should be used, when possible, for study, since many of the finer shadows are lost in the printing.

Old calcified tubercles give the most decided shadow, and lesions of this kind can be recognized as small as 3 mm. in diameter (Plate XIII, A and B).

Old scars or fibrous tissue cast a less dense shadow, and require larger lesions, but they can usually be recognized by their bandlike appearances (Plate XIII, B).

Consolidations vary in the density of their shadows with the size of the lesion, but an area 1 cm. in diameter can be recognized in an emaciated person (Plate XII, B, and XIII, A). Consolidations seldom occur singly, therefore they give the lung a mottled appearance (Plate XIV, A and B), except when there is massive consolidation (Plates XIV, B, XV, A and B, XVI, A). Finally the consolidation can be recognized by comparison of the suspected area with that of the opposite lung, or with the other parts of the same lung. The tuberculous deposits, or the affected areas lying in different planes, are thrown upon the plate in one plane. Therefore, upon superficial observation, one might conclude that a greater proportion of the lung is affected than is correct. That is, the whole lung might show a mottled appearance, and the false conclusion be drawn that no healthy lung tissue remained. This mistake need not be made by an experienced observer, because, if the whole lung or lobe is affected, the degree of general density will be much greater than when only scattered areas are involved, and the density will approach that of the shadow of the heart (Plates XIV, B, XV, A and B, XVI, A). Even when a whole lobe or lung is involved, the shadow is rarely uniformly dense, because the lung is rarely uniformly consolidated (Plate XVI, A). There are likely to be small or large areas of cavity or compensatory emphysema associated.

Cavities are usually recognized by their increased transparency, surrounded by the shadow of consolidation (Plates XVI, B, and XVII, A). If the cavity is large, there will be little difficulty in recognizing it. If, however, the cavity is small, or is resting upon a large area of consolidation or thickened pleura, it is less easily recognized. Under favorable circumstances, a cavity 1.5 cm. can be recognized.

Emphysematous areas are also noticeably transparent. A dense shadow may be found on one side of this area, but it is likely to have the other side continuous with more or less healthy tissue, and therefore can be differentiated from a cavity (Plates XV, B, XVII, A, XVIII, A).

Pulmonary Abscess.—This diagnosis cannot be entirely made by means of the Röntgen ray, but when it is suspected it can be more accurately localized by the assistance of the rays. The radiographic appearances will not differ materially from those of a cavity, except that the surrounding area of consolidation is likely to be larger in proportion to the size of the cavity (Plates XVII, B, and XVIII, A). Since the two may be associated, however, the difficulties are increased, and the findings must be carefully compared with the physical signs before operating.

Pulmonary Gangrene.—The remarks made above in connection with pulmonary abscess will apply to pulmonary gangrene, except that, instead of the area of consolidation being large and the cavity small, the cavity is more likely to be large and the surrounding wall small. The conditions and the shadows will vary with the stage at which the examination is made. The odor will usually suggest the diagnosis, and the rays will be useful in locating the area for operation (Plates XVIII, B, and XIX, A).

Pneumonia.—The fluoroscope shows a dense shadow in the affected area, and the movements of the diaphragm are diminished on the affected side, partly because of the increased density of the lung and partly at times because of an adhesive pleurisy. Williams was one of the first to apply the rays in the study of pneumonia. He depended almost entirely upon the fluorescent screen. It is also a most useful adjunct in the diagnosis of lesions not centrally located, especially when such lesions are situated beneath the scapula, or when small consolidations are surrounded by areas of compensatory emphysema. Compensatory emphysema assists in outlining the lesions by means of the Röntgen ray because of its greater transparency, but offers considerable hindrance when physical signs are depended upon. De la Camp found even in lobar pneumonia that the whole lobe is seldom uniformly consolidated, and he often found by means of the rays extension of the process in another lobe or in the opposite lung, when ordinary physical examination failed to reveal such lesions.

He also found the rays of great assistance in the study of the cases during resolution. In some cases, months were required for the lung to completely recover. The rays show lesions long after the physical signs are negative. In one case, in which the patient complained of pain in the region of the heart, which persisted four months after the crisis and after all physical signs were negative, the Röntgen examination showed a fibrous band, about 0.5 cm. broad, binding the left side of the diaphragmatic pleura to the pericardial sac. Other symptoms of obscure origin following pneumonia may often be cleared up by the aid of a Röntgen examination. The same principles that have been described in the interpretation of tuberculous negatives will apply to those of pneumonia. The areas involved, however, are usually larger, and therefore more easily recognized (Plate XV, A).

Emphysema.—In a typical case of emphysema, one is struck by the great transparency, which is greater than in any other condition of the lungs. This transparency affects both lungs. The ribs will be found to extend outward from the spinal column at more nearly a right angle than normal. To distinguish the lesser grades of emphysema, or to recognize local areas, much more experience is necessary. For then one must keep in mind the normal Röntgenogram of a patient of the same age and general development, thickness of the chest walls, etc., and made under similar conditions (see Compensatory Emphysema).

Collapse of the Lung.—Collapse of the lung will probably not be recognized by means of the Röntgen rays when the area involved is small, but when the area is large it gives an appearance very similar to consolidation. It is more uniform and more sharply outlined than tuberculosis, and it involves a lesser area than when pneumonic consolidation is present.

THE PLEURA

Thickened Pleura.—Thickening of the pleura is a common affection, occurring both independently and in association with tuberculosis of the lung. This condition is recognized in the Röntgenogram as a uniform shadow of only slight density. The density will vary with the degree of thickening. This shadow is then seen to shade gradually at its edge into the surrounding clear space. If the surrounding lung is healthy, there will be little difficulty in recognizing a moderate thickening. If, however, the thickened pleura is overshadowed by consolidation of the lung, it becomes more difficult, and at times impossible. Here experience alone will serve as a guide. In order to study the pleura accurately, both an anterior and posterior plate should be made (Plates XIX, B, XIII, A, XVII, A).

Pleural Effusion.—Pleural effusion is best recognized by placing the patient in the erect posture, with the plate or screen posteriorly and the tube anteriorly. To make a Röntgenogram: Place the patient in a chair, with a large board resting against the back, and upon which the plate is supported. The pleural effusion gives a uniform but not very dense shadow, occupying the lower part of the pleural cavity. The upper level of the shadow has a curved, but not an irregular, line. In uncomplicated effusion, the shadow is even more uniform than that of a thickened pleura. Pleural thickening will also be recognized by its irregular border, which is never a line. The shadow is less dense than would be produced by the consolidation of the lobe of lung (Plate XX, A).

Pneumothorax is recognized by the area of great transparency (greater than in emphysema). This area is elongated in a vertical direction, as a rule, and occupies the lower lateral portion of the chest. Toward the median line may usually be seen the thickened pleura and the diseased lung. The heart is commonly displaced to the opposite side (Plate XX, B).

Hydropneumothorax is more common, and forms one of the most interesting fluoroscopic pictures that can be found. In addition to the findings of pneumothorax, the fluid at the base of the pleural cavity may be seen to move with each respiration or each movement of the body. If the patient is shaken, the fluid is seen to splash (Plate XX, B).

Hemothorax.—I have examined two cases of this character. Both showed a more dense shadow than would be produced by any other form of pleural effusion (Plate XXI, A).

Consolidation and Pleural Effusion Combined.—Plate XX, A, shows a case of this character. The shadow of the pleural effusion may be seen to extend to the fifth rib on the right side, and to the seventh rib on the left side. The consolidation of the right middle lobe shows through the effusion, and is distinctly more dense. Probably no condition will give more difficulty in recognition than consolidation surrounded by an extensive pleural effusion. In this particular case we had that peculiar physical sign, viz., tubular breathing and bronchophony extending throughout the area of pleural effusion. By making Röntgen examinations in all such cases, we may find that consolidation is present, and that it is the cause of the peculiar sign. This sign is probably due to the transmission of the larger sound-waves from the bronchi, through the consolidated lung to the fluid in the pleural cavity, and thence through the chest wall to the ear. The striking of two stones together under water will convince most people that fluids will transmit sound, but these sounds must be of the louder and coarser variety; therefore, vesicular breathing is not transmitted through an effusion.

Subphrenic Abscess.—The difficulty in recognizing many of these cases is appreciated by all who have had much experience, and therefore any aid will also be appreciated. In a case examined about two years ago, by means of the screen I was able to see a decided displacement of the diaphragm upward as far as the ninth rib, and absolute immobility on the right side. The left side of the diaphragm was seen to move 2½ in. (12.5 cm.). The Röntgen examination will also eliminate some of the diagnoses with which it may be confused, such as pneumothorax, encysted pleural effusion, or empyema (Plate XXI, B).

THE HEART

Normal Size and Position.—This is given under the description of the Röntgenogram of the normal thorax, to which the reader is again referred (Plate XII, A). Unfortunately, our technic has not been so far perfected as to enable us to photograph the heart in a part of its cycle, which would necessitate an exposure of less than a second. A few Röntgenograms of the heart have been made in a fraction of a second (Plate XXII, A), but this has not yet become a working method, for only by the most perfect technic and powerful apparatus can this be accomplished.

Cardiac Hypertrophy.—An enlargement of the heart can be more certainly and accurately recognized and recorded by means of the Röntgen rays than by any other method. Not only the general enlargement, but an enlargement of any of the four cavities can be shown in the Röntgenogram. In cardiac hypertrophy the enlargement is most likely to involve either the right or the left ventricle, or both (Plates XXII, B, and XXIII, A).

Cardiac Dilation.—The Röntgenogram of cardiac dilation differs from the appearances of cardiac hypertrophy in that the enlargement extends not only laterally, but also in the vertical direction, showing enlargement of the auricles, and the whole cardiac shadow being more globular (Plate XXIII, B). The fluoroscope, in this condition, shows beautifully the waves of contraction.

Cardiac Displacement.—The heart may be displaced by a number of conditions, each of which can be determined by means of the Röntgen ray. Dextrocardia may be due to pneumothorax, as is shown in Plate XX, B, or by fibrous bands occurring in the course of tuberculosis, as is shown in Plates XXIV and XXV, A. (See also Plate XXV, B.)

Pericardial Effusion.—The shadow of pericardial effusion differs only slightly from that observed in cardiac dilation, but it is more triangular; the apex of the triangle is more acute, and by Röntgenoscopic examination the waves of pulsation are much less distinct.

Aneurism.—While the Röntgen examination should not be the only method, surely no other is more reliable in making a diagnosis. The shadow will vary with the size, form, and location of the aneurism, but certain characteristics will remain. The tumor mass will involve the line of the aorta, or the great vessels. It will give a more uniform shadow, instead of the localized areas which characterize groups of enlarged mediastinal glands. By means of the fluoroscope.

the shadow will usually show expansile pulsation. If, however, the aneurism has thick or inelastic walls, such as occurs in a healed aneurism, this symptom will be absent. By means of the fluoroscope, the aneurism should be examined from various directions, so that if any pulsation be present it will not be overlooked (Plate XXVI, A and B).

Mediastinal Tumors.—The symptoms will almost always lead us to suspect some pressure in the mediastinum; but since these symptoms may be due to a number of conditions, the diagnosis is often difficult, and the Röntgen examination will be found most valuable.

RÖNTGEN EXAMINATION OF THE GASTRO-INTESTINAL TRACT

THE STOMACH

Occasionally, in thin people, the stomach may be seen naturally, because it is distended with gas. As a rule, however, artificial assistance must be supplied. One method is to distend the stomach with gas, which is best done by means of an atomizer bulb or household syringe attached to a stomach tube. This is sometimes advisable when a tumor of the adjacent viscera is under consideration, because the lighter area due to the gas may be distinguished from the shadow of the tumor. Such examinations, however, are only possible under favorable circumstances—e. g., when the tumor is of considerable size and the patient thin. Generally, inflation of the stomach with gas is bad practise, and unsatisfactory.

A far better method is to render the stomach opaque by means of an emulsion of bismuth subnitrate—about one ounce to the pint of milk. This method has been used successfully by Williams, Cannon, Rieder, and by myself. By this means the size, form, position, and the motility of the stomach may be determined. In these examinations, the plate will be found much more useful than the screen because of the more delicate shadows obtained, and because it will form a permanent record.

THE INTESTINES

The examination of the intestines is really only a continuation of the examination of the stomach, providing plates 14 × 17 in. are used. Where no interest is attached to the study of the stomach the plates need not be made until twelve hours after the ingestion of the food, unless an obstruction in the small intestines is suspected. Then the examination should be made in at least six hours. By this means the rate of movement and the position of the large bowel can be determined. The position of the large bowel, and particularly the transverse colon, can be determined by giving the emulsion of bismuth by enema.

THE LIVER

In children, the outline of the liver, both its upper and lower border, can be determined, but in adults the lower border is usually indistinct. The upper border in adults can usually be studied, except where the shadow of the heart interferes. The affections involving the upper border of the liver will be shown in the changes in the curve and position of the diaphragm. These affections may be abscess, subphrenic abscess, gummata, neoplasms, echinococcus cysts, etc.

Gall-stones have been recognized in a few instances, but the Röntgen examination is not yet reliable as a diagnostic factor, except as possibly confirmatory evidence.

THE SPLEEN

The Röntgen rays give little assistance in the study of this organ. In children it may be observed, and in adults the lower border can be seen if the spleen is large, or if the intestines are distended with gas.

THE KIDNEYS

Examination of the kidneys directly will give little additional information. The outline of the kidney can only be seen in children, in thin adults, or when the kidney is much enlarged.

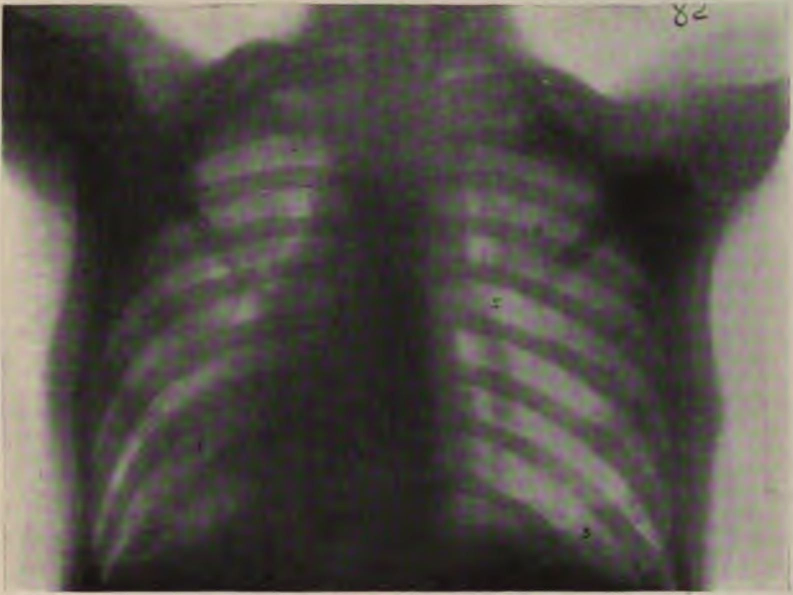
Floating kidney can very rarely be observed, and then ordinary methods of physical examination will determine the condition more accurately and more certainly.

In general, the diagnostic value of the evidence obtained through the Röntgen ray will depend very much upon the skill and experience of the operator both in Röntgen work and in general medicine. The ability to read a negative will also depend in great part upon the physician's knowledge of general medicine.

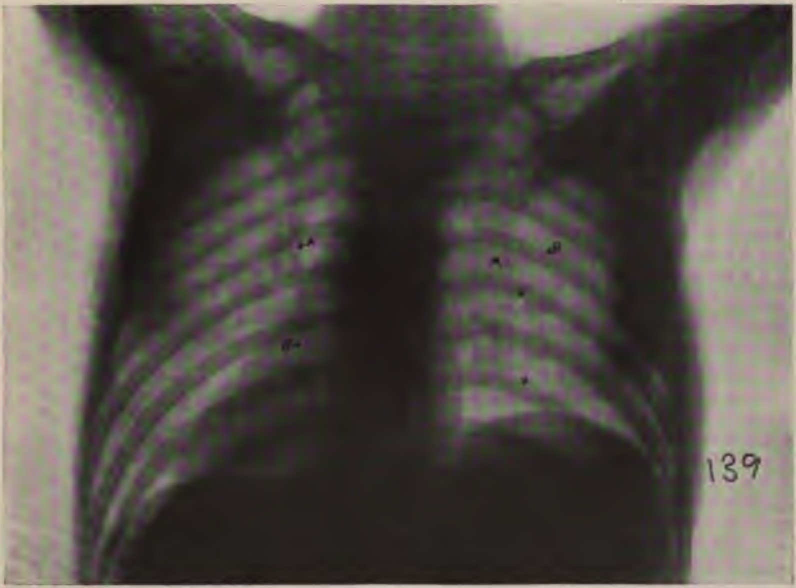
PART IV

RÖNTGENOGRAMS

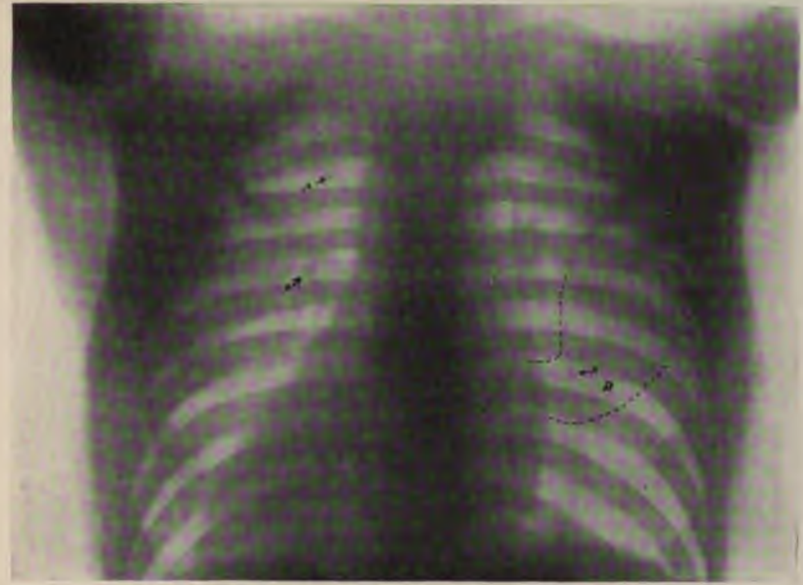
PLATE XII



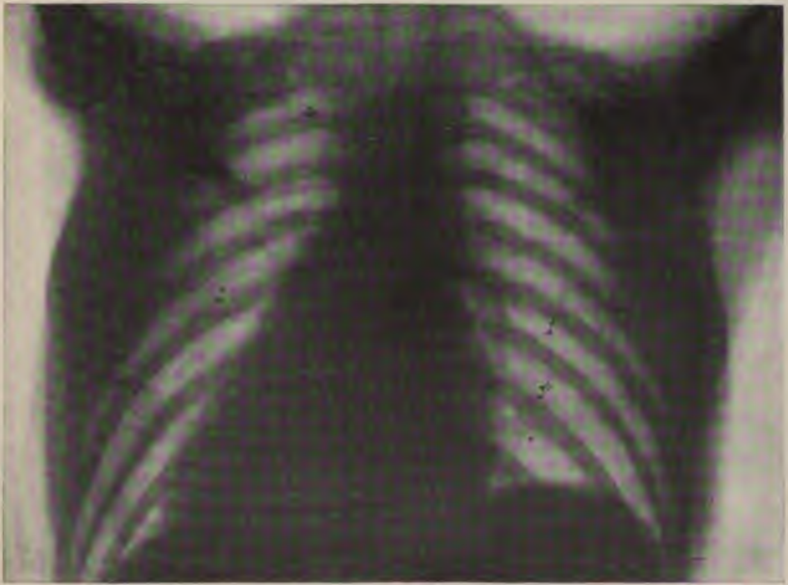
A.—Normal Thorax of a Young Man. (1) Shadow of the Heart. (2) Shadows of the Large Pulmonary Vessels. (3) Three Lines of Shadow of the Diaphragm. Exposure, Fifteen Seconds.



B.—Incipient Tuberculosis, Showing the Earliest Possible Lesions. (*A*) Areas of Congestion, as Determined by Autopsy. (*B*) Isolated Tubercles, the Size of a Pinhead. (*C*) Branching Pulmonary Vessel.

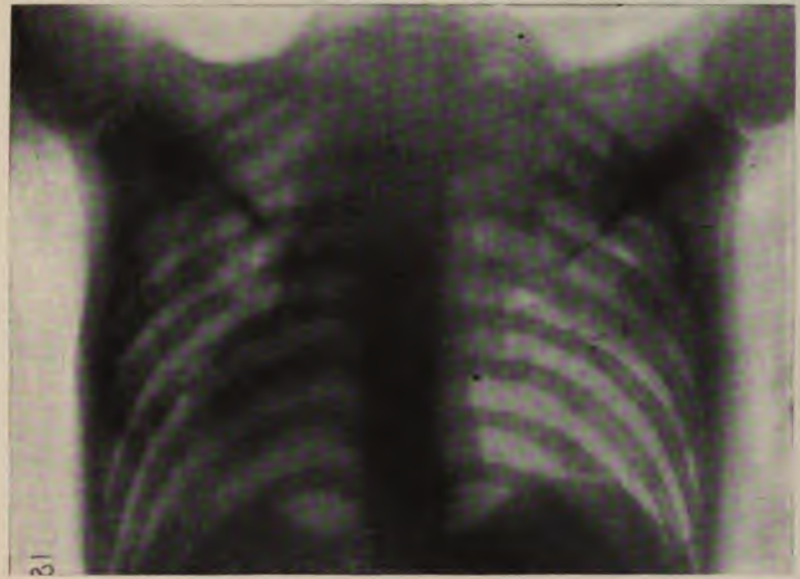


A.—Shows Infiltration of Both Apices with Scattered Deposits Lower Down, and Dense Shadows Probably Indicating Calcified Tubercles (*A*) Also a Uniform Shadow at the Angle of the Right Scapula which Probably Indicates a Thickened Pleura.

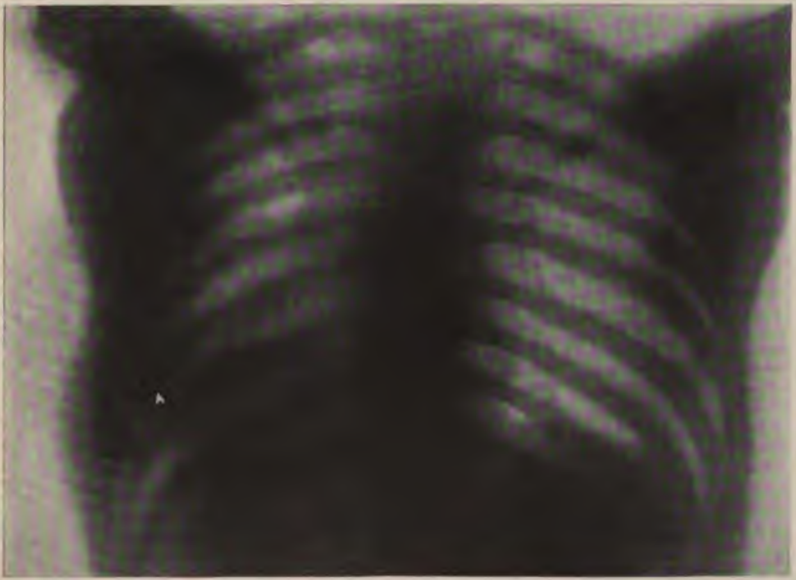


B.—Shows Small, Rather Dense Fibrous Bands at the Apices of Both Lungs —Old Fibrous or Healed Lesions. (*AA*) Old Calcified Tubercles. (*B*) Old Fracture of Ninth Rib. (*D*) A Large Deposit at the Left Apex.

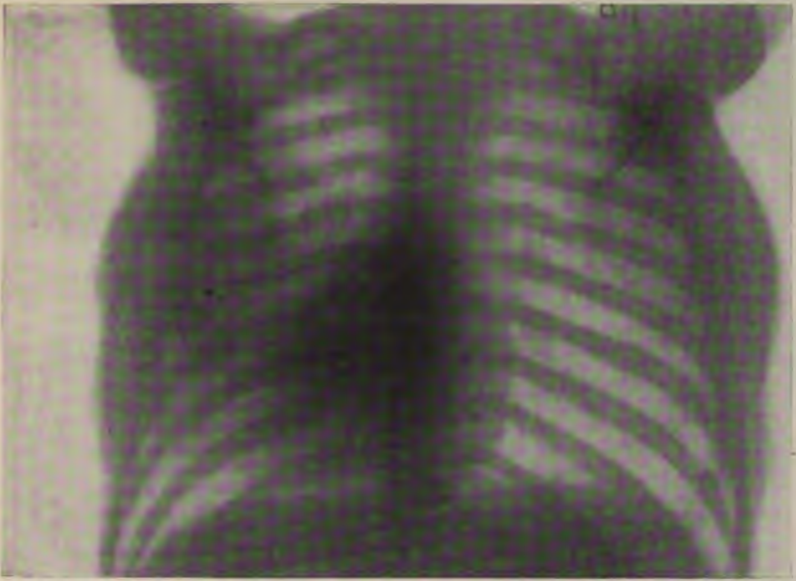
PLATE XIV



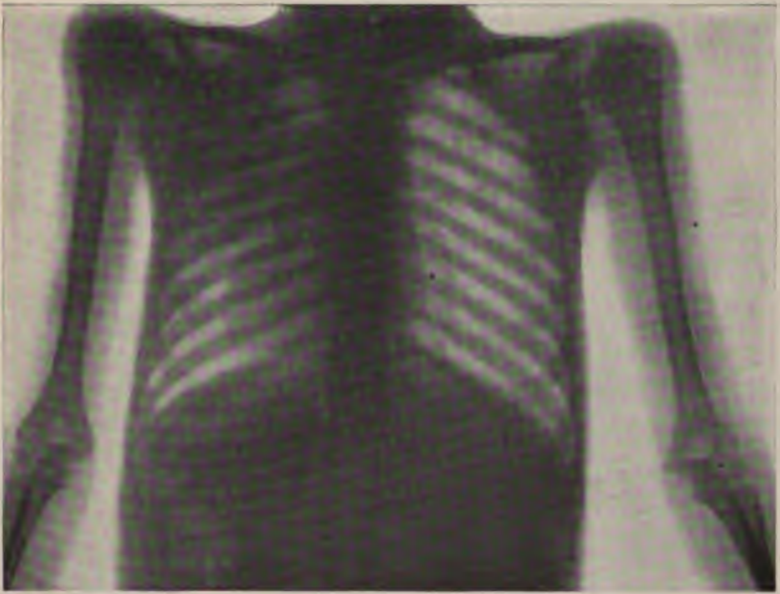
A.—General Infiltration (Acute Process) of Both Lungs with No Large Areas of Consolidation or Cavitation.



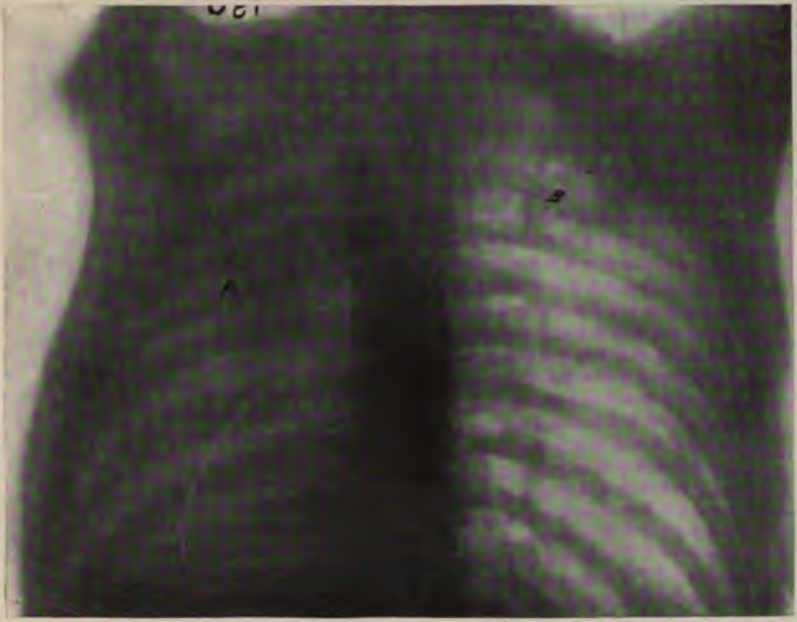
B.—General Infiltration, with a Larger Area of Consolidation to the Left of the Apex of the Heart (*A*).



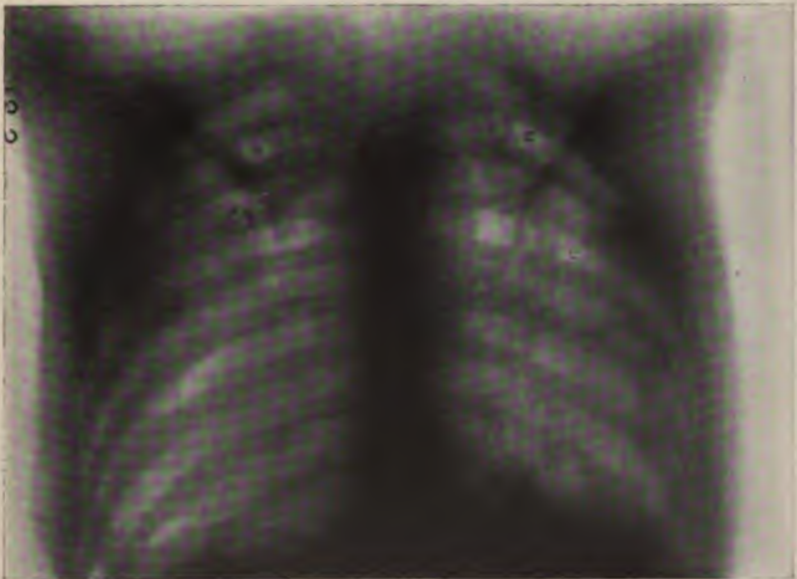
A.—A Large Area of Complete Consolidation Lying to the Left of the Heart with Small Infiltration Projecting Toward the Apex. This Followed an Abortion and Was at First Thought to Be an Infarct.



B.—A Nine-year-old Child. Posterior View. Consolidation of the Lower Portion of the Upper Lobe of the Left Lung, with Compensatory Emphysema of the Lower Lobe.

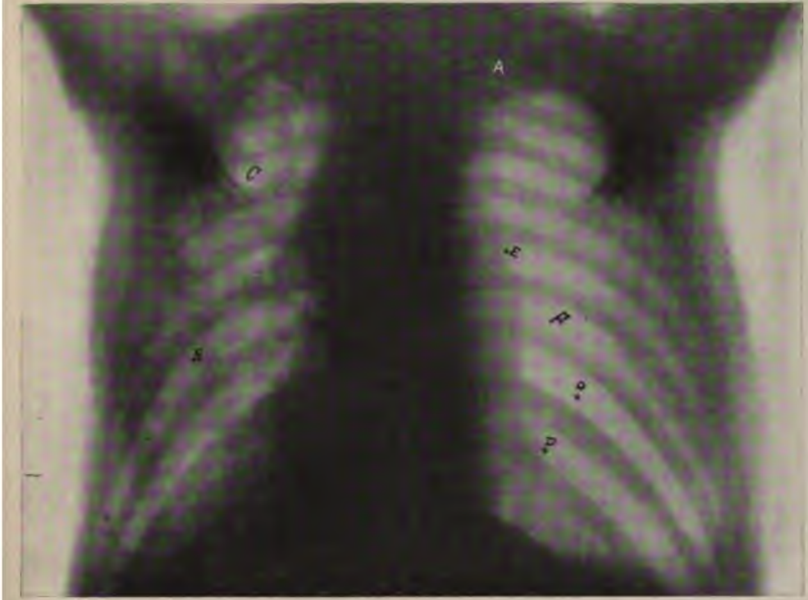


A.—(*A*) Consolidation of the Left Lung of Marked but Not Uniform Density. The Outline of the Heart Is Indicated by the Dotted Line. (*B*) Scattered Tuberculous Deposits in the Right Lung.

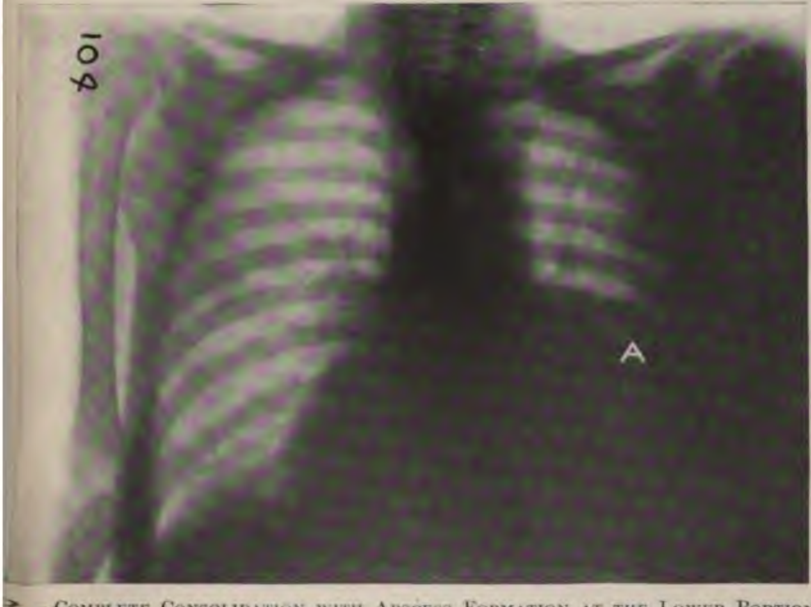


B.—General Infiltration of Both Lungs, with at Least Two Cavities in the Right Lung and One in the Left (*C*, *C*, *C*).

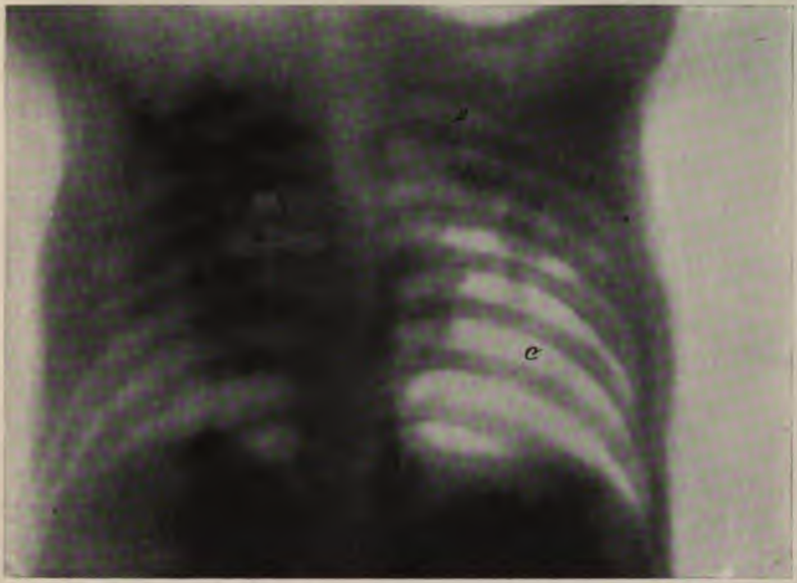
PLATE XVII



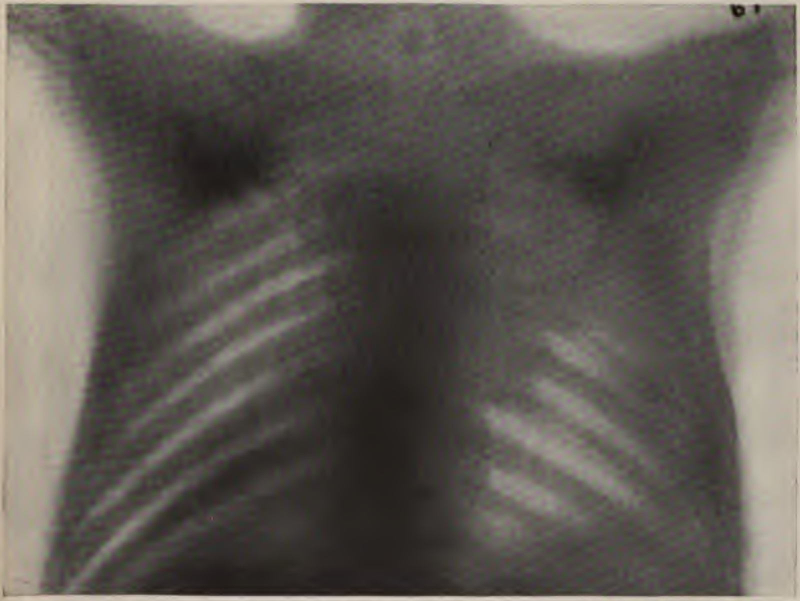
A.—(*A*) Consolidation at the Right Apex. (*B*) Scattered Tuberculous Deposits in the Base of the Left Lung. (*C*) Large Cavity in the Left Apex, Subdivided by Fibrous Bands. (*D, D*) Probable Old Fractures of the Ribs with Thickened Pleura Lying Beneath. (*E*) Dilation of the Ascending Aorta. (*F*) Compensatory Emphysema of the Right Lung.



B.—Complete Consolidation with Abscess Formation at the Lower Portion of the Right Lung (*A*); Tuberculous Infiltration of the Left Lung.

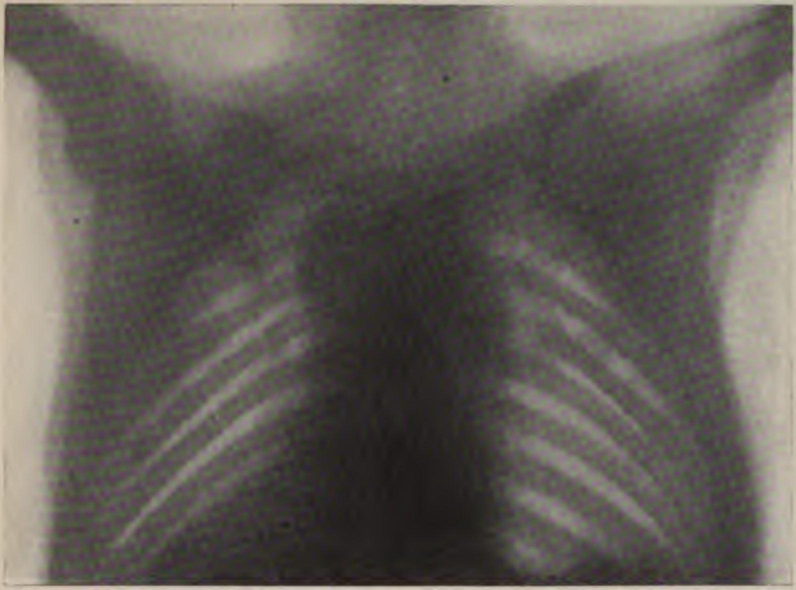


A.—(*A*) ABSCESS OF THE LEFT LUNG, SURROUNDED BY TUBERCULOUS CONSOLIDATION. (*B*) CONSOLIDATION AT THE RIGHT APEX, WITH (*C*) COMPENSATORY EMPHYSEMA BELOW.

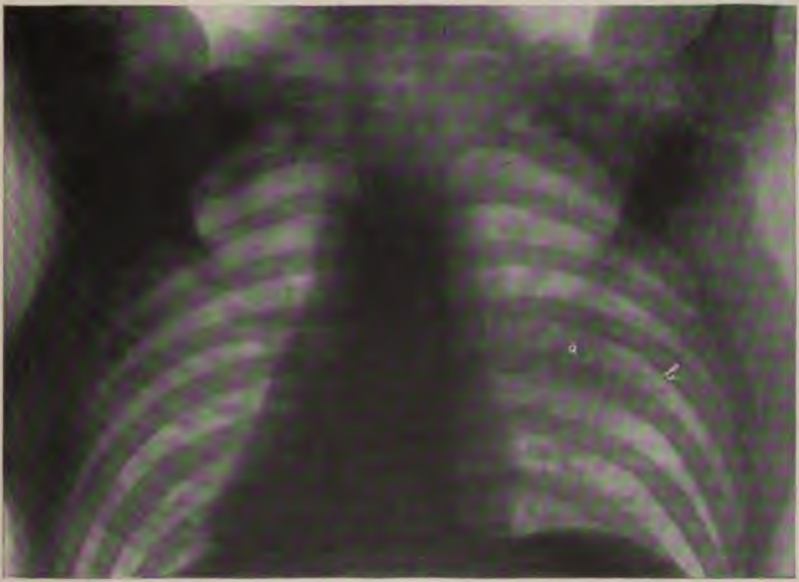


B.—GANGRENE OF THE LUNG (DOTTED LINE). DOUBLE SHADOWS DUE TO COUGHING WHILE UNDER EXAMINATION.

PLATE XIX

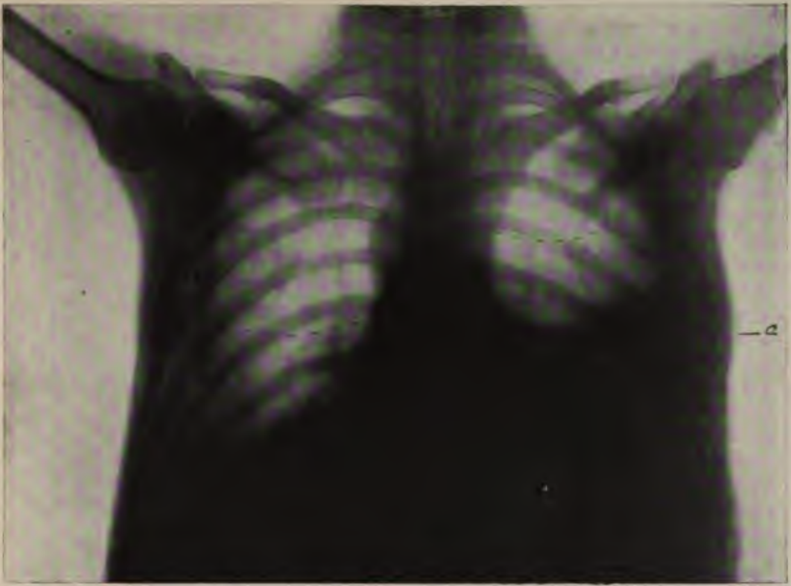


A.—Same Case as Plate XVIII, B, After Operation. Absence of Portion of Resected Fifth Rib Shown; Also of the Shadows at the Right Apex.

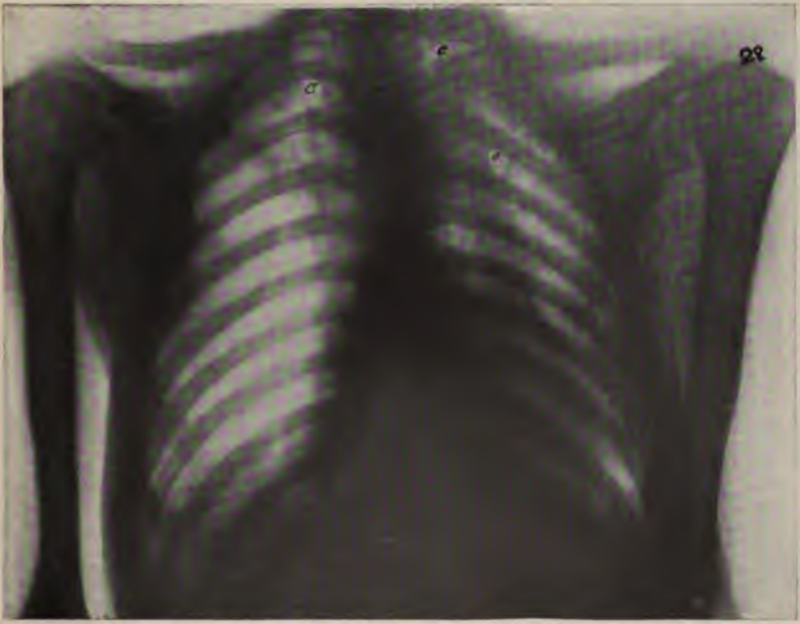


B.—General Small and Probably Early Infiltration, with a Thickened Pleura (*a*) and a Calcified Tubercle Near the Angle of the Right Scapula (*b*).

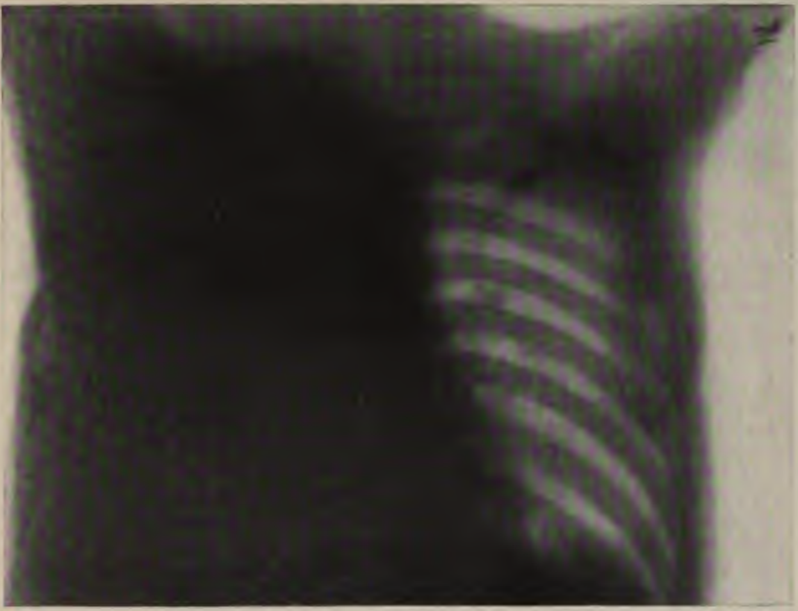
PLATE XX



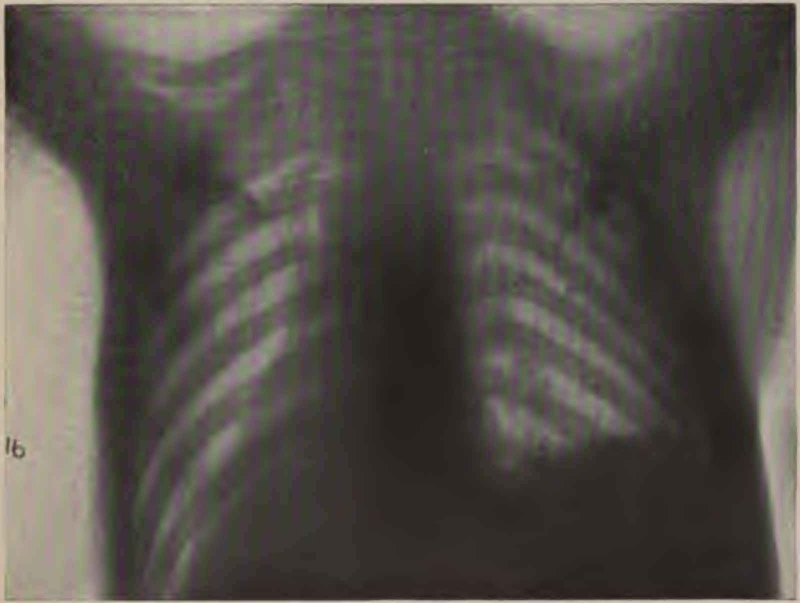
A.—Pleural Effusion Extending to the Fifth Rib Posteriorly on the Right Side and to the Seventh Rib on the Left Side; Also a Consolidation on the Right Side in the Region of the Right Middle Lobe. (*c*) Compensatory Emphysema Above the Effusion.



B.—Light Area on the Left Side Due to Pneumothorax. Shadow of Fluid at the Bottom, About Two Inches Above the Diaphragm. Compression of the Left Lung with Cavities at the Apex (*C*, *C*, *C*). Heart and Aorta in the Right Chest. Tuberculous Deposits in the Right Lung with Cavity at the Apex (*C*).

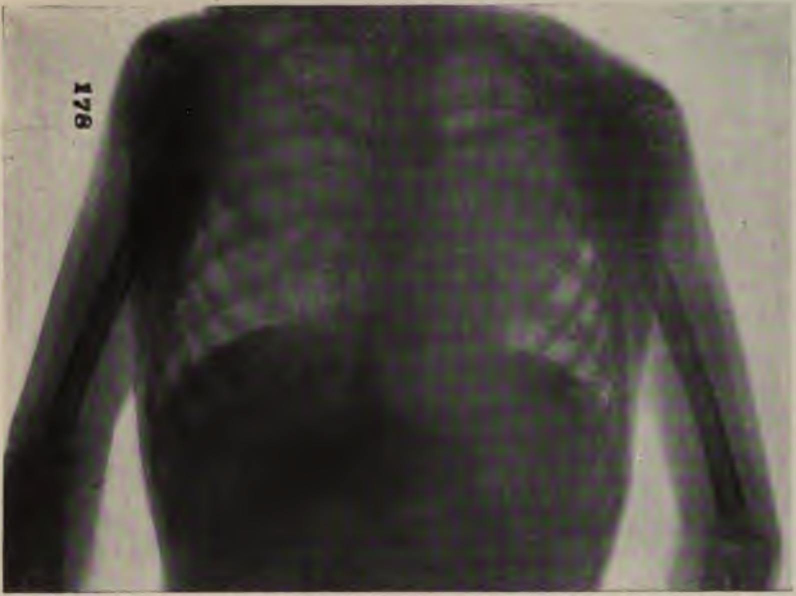


A.—Hemothorax on the Left Side of the Chest, Displacing the Heart to the Right.

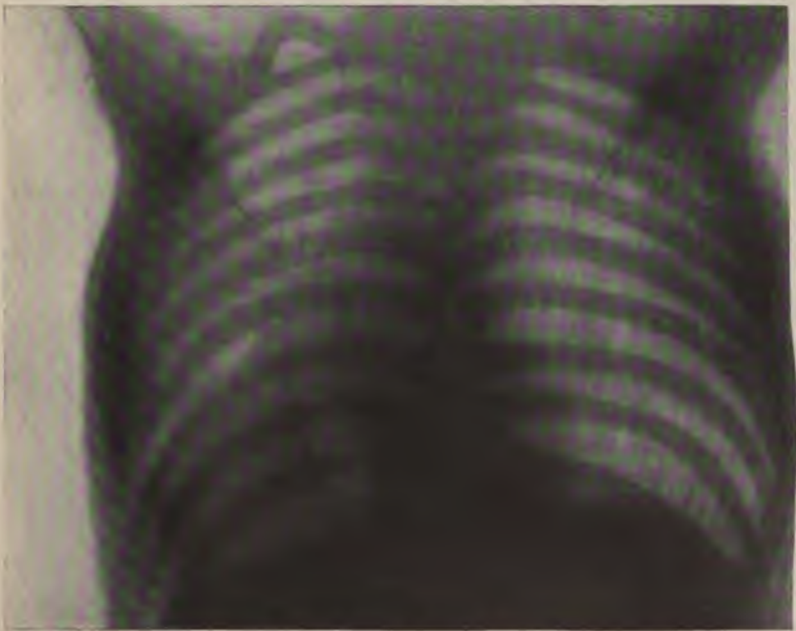


B.—Subphrenic Abscess of the Right Side. Diaphragm Displaced Upward One and One-half Inches.

PLATE XXII

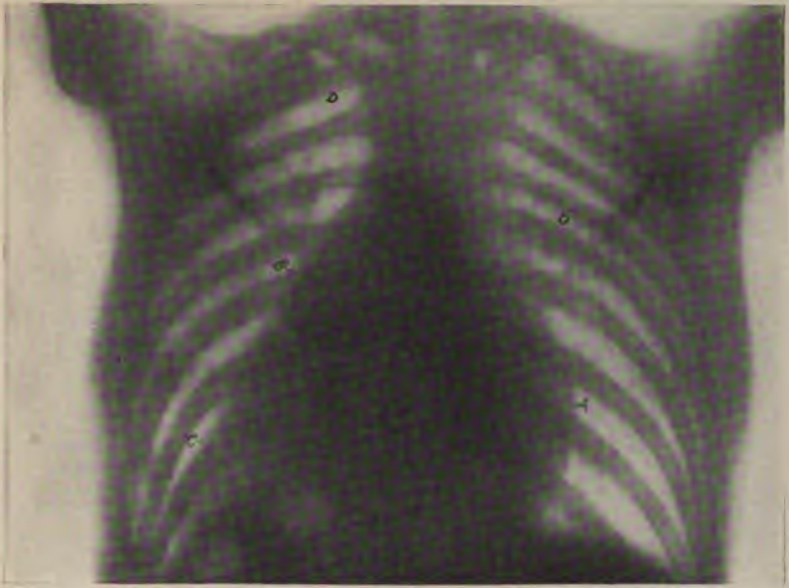


A.—A NINE-YEAR-OLD CHILD. ANTERIOR VIEW. CONSOLIDATION OF THE LOWER PORTION OF THE LEFT UPPER LOBE.

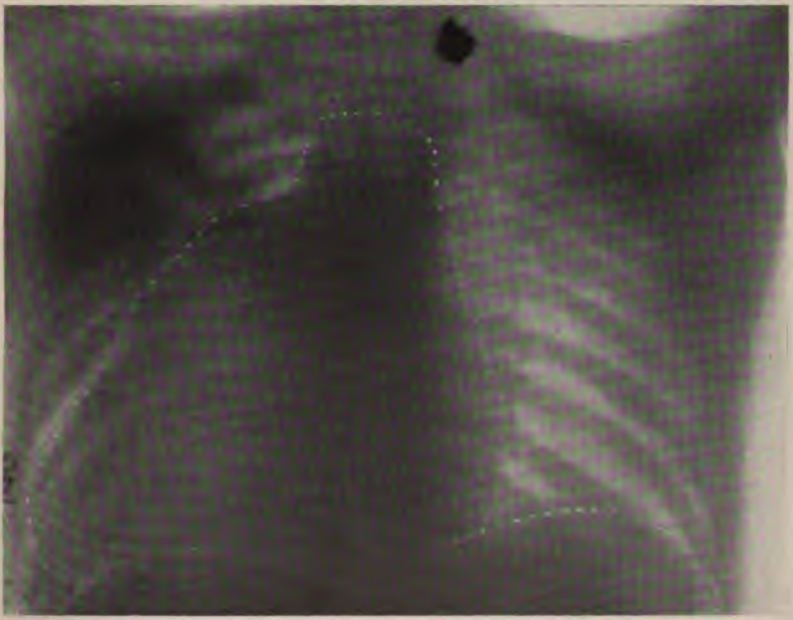


B.—MARKED HYPERTROPHY AND DILATION OF THE LEFT VENTRICLE. CONGESTION OF THE BASE OF THE RIGHT LUNG. SOME ENLARGEMENT OF THE WHOLE HEART.

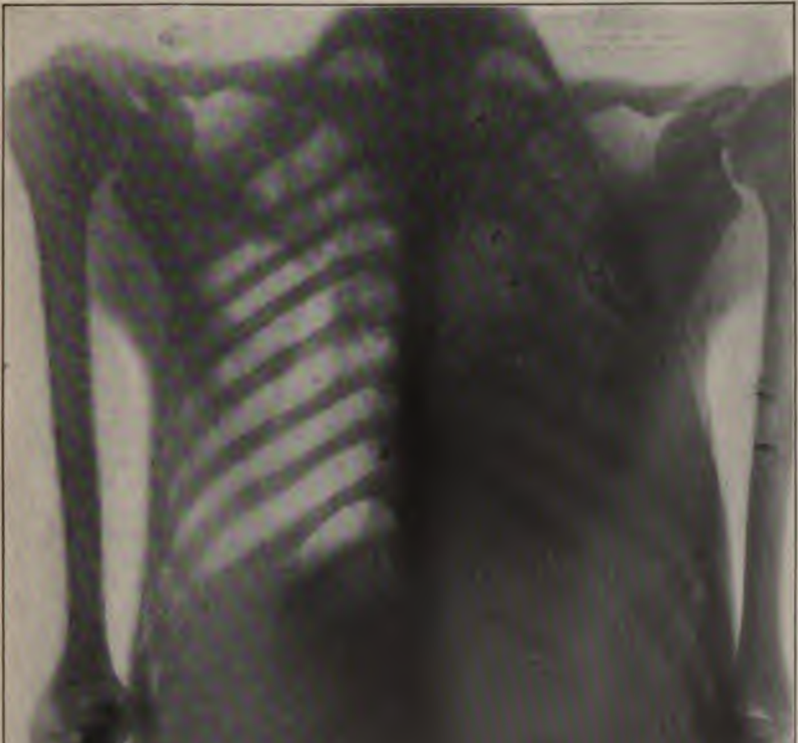
PLATE XXIII



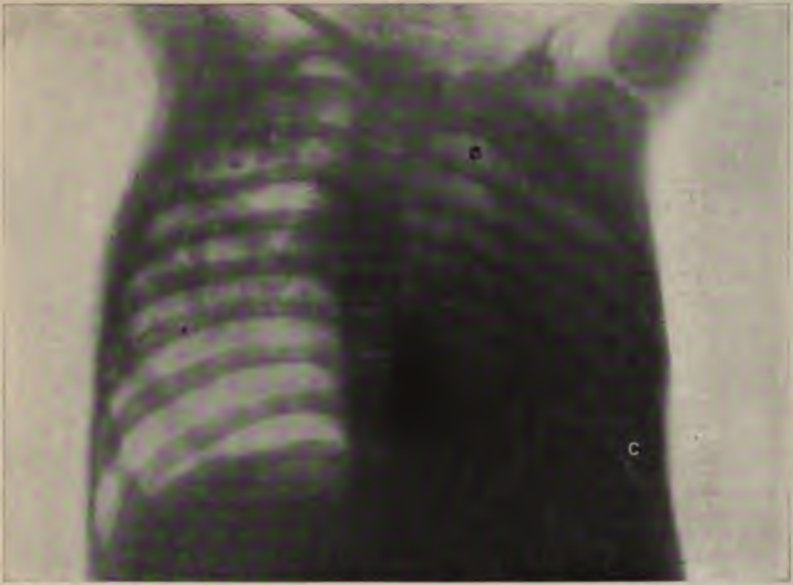
A.—(*A*) Hypertrophy of the Right Ventricle. (*B*) Hypertrophy of the Left Auricle. (*C*) Left Ventricle, Nearly Normal in Size. (*D*, *D*) Probable Tuberculous Deposits.



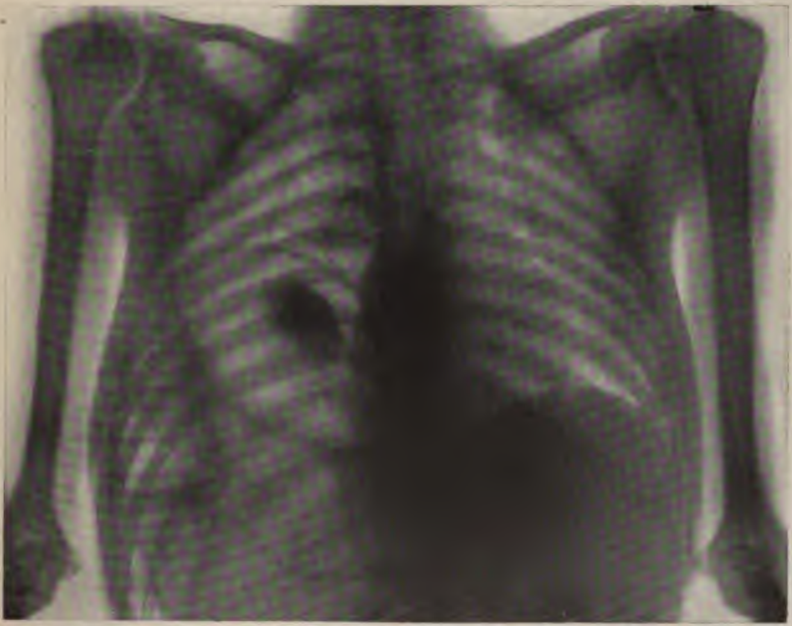
B.—Marked Cardiac Dilation. Note the Globular Appearance of the Heart, and the Enlargement Upward of the Left Auricle. The Dotted Lines Indicate the Arch of the Aorta, and the Diaphragm, also.



Dextrocardia Due to Tuberculosis. Complete Consolidation of the Right Lung, with Five Cavities at the Apex (Light Areas Surrounded by Dense Shadows). Compensatory Emphysema of Left Lung. Tuberculous Infiltration at Left Apex.

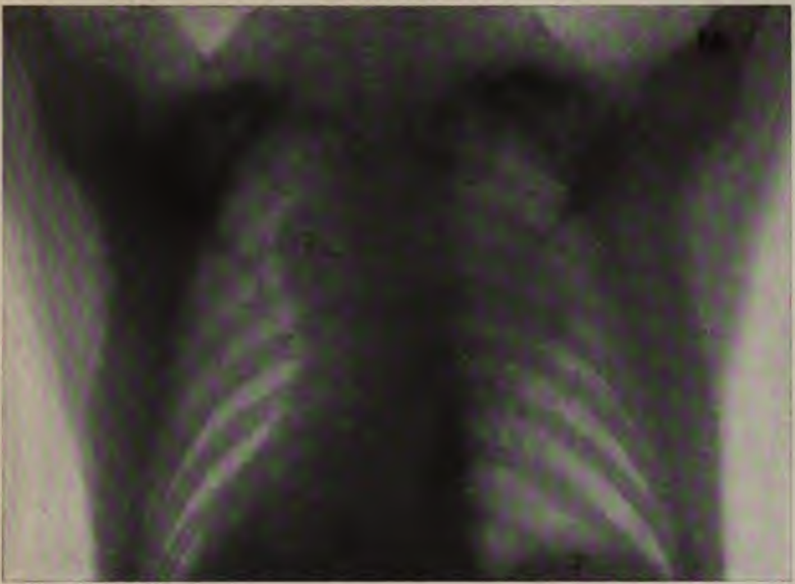


A.—Dextrocardia. Tuberculous Deposits in the Left Apex, with Compensatory Emphysema Below. Complete Consolidation of Right Lung, with a Cavity at the Apex, and Another at the Base (*c*, *c*).

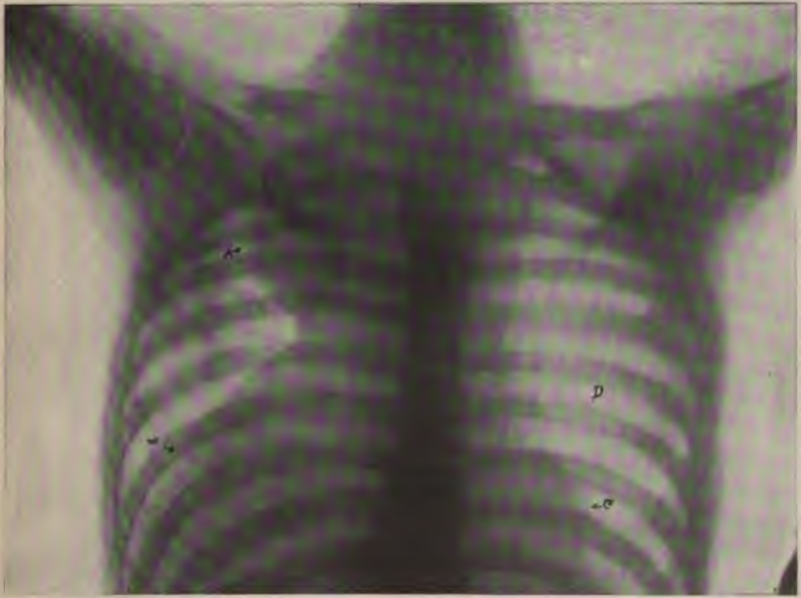


B.—Shows: (1) the Left Side of the Diaphragm Four Inches Higher than the Right. (2) The Heart Displaced to the Right. (3) The Outline of the Stomach, Below the Diaphragm, Containing a Bolus of Food. (4) The Descending Colon, Showing the Transverse Folds. (5) Shadows of Congestion of the Lungs.

PLATE XXVI

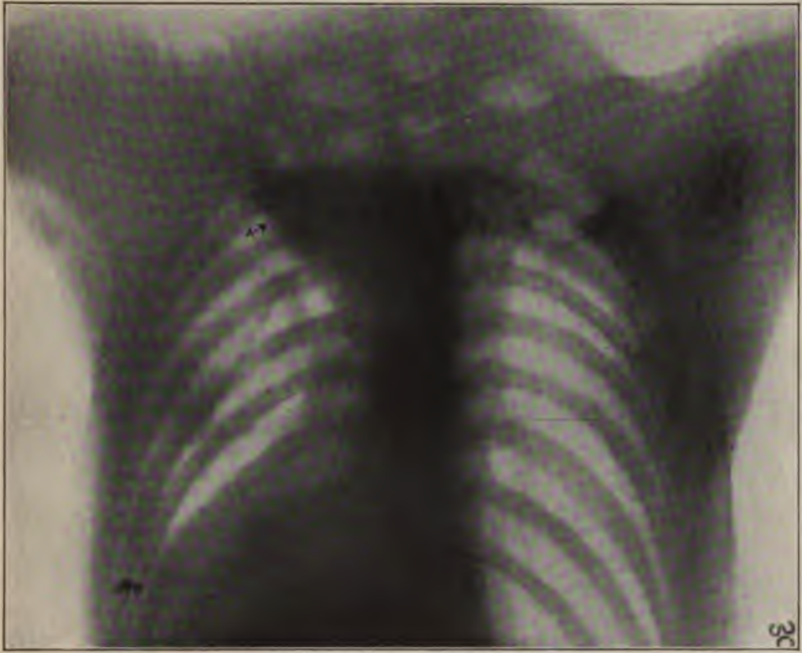


A.—Aneurism of the Arch of the Aorta, Involving Chiefly the Descending Portion. The Heart Is Displaced Downward and to the Left.

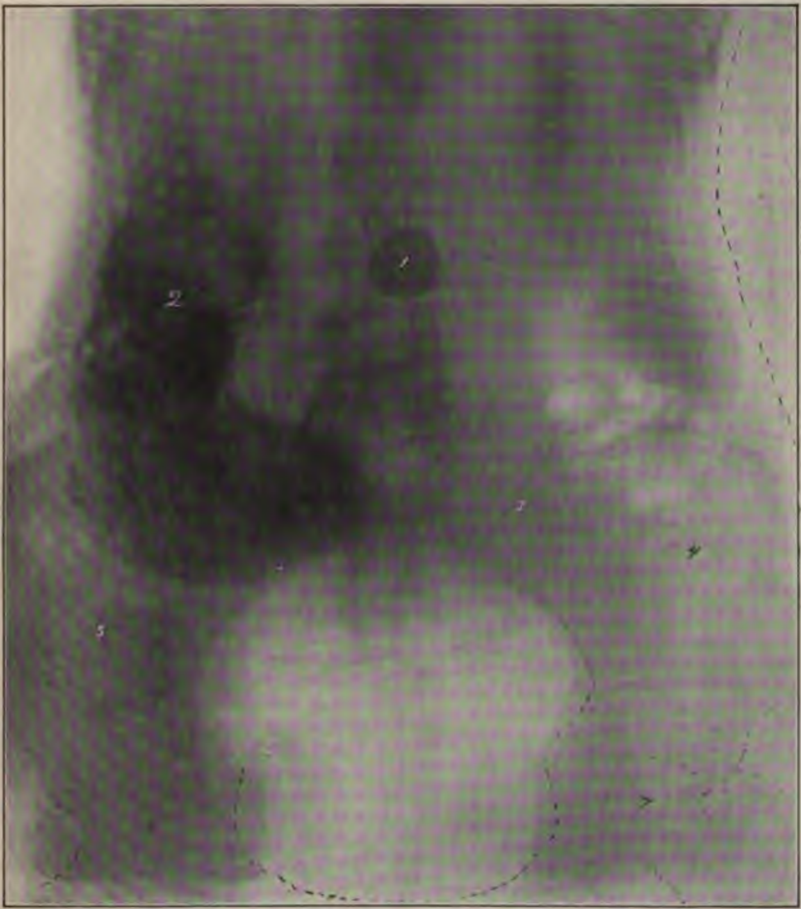


B.—(*A*) Aneurism of the Descending Aorta. Some Erosion of the Third, Fourth, and Fifth Dorsal Vertebræ Is Shown. (*B*) Enormous-Enlargement of the Left Ventricle. (*C*) A Second Aneurism, or an Enlarged Right Ventricle. (*D*) Compensatory Emphysema of the Right Lung.

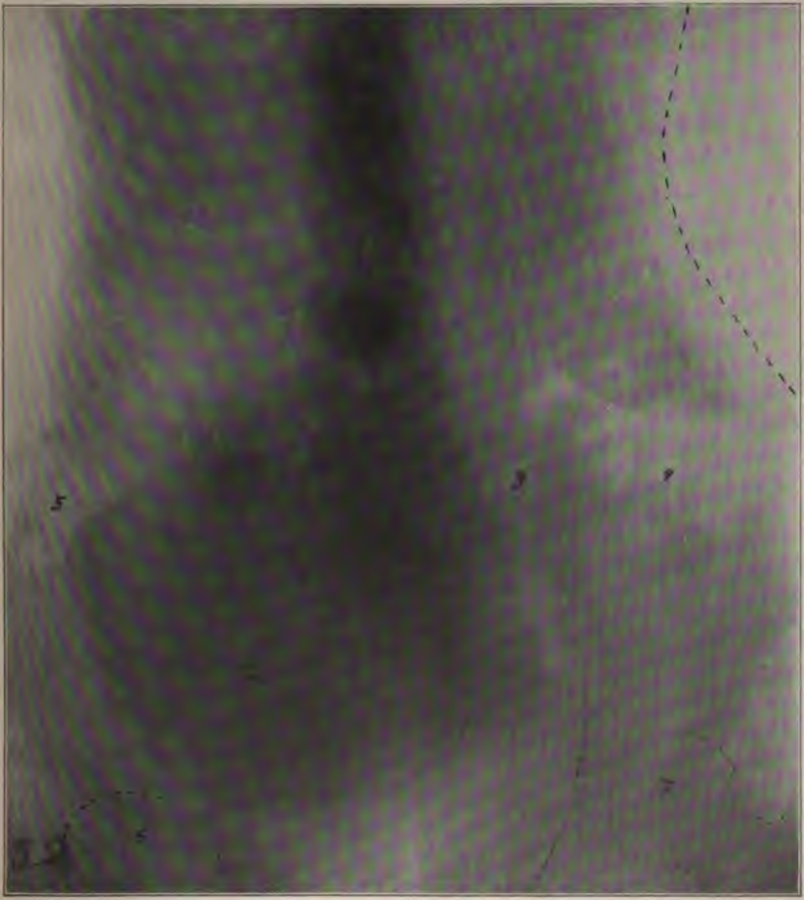
PLATE XXVII



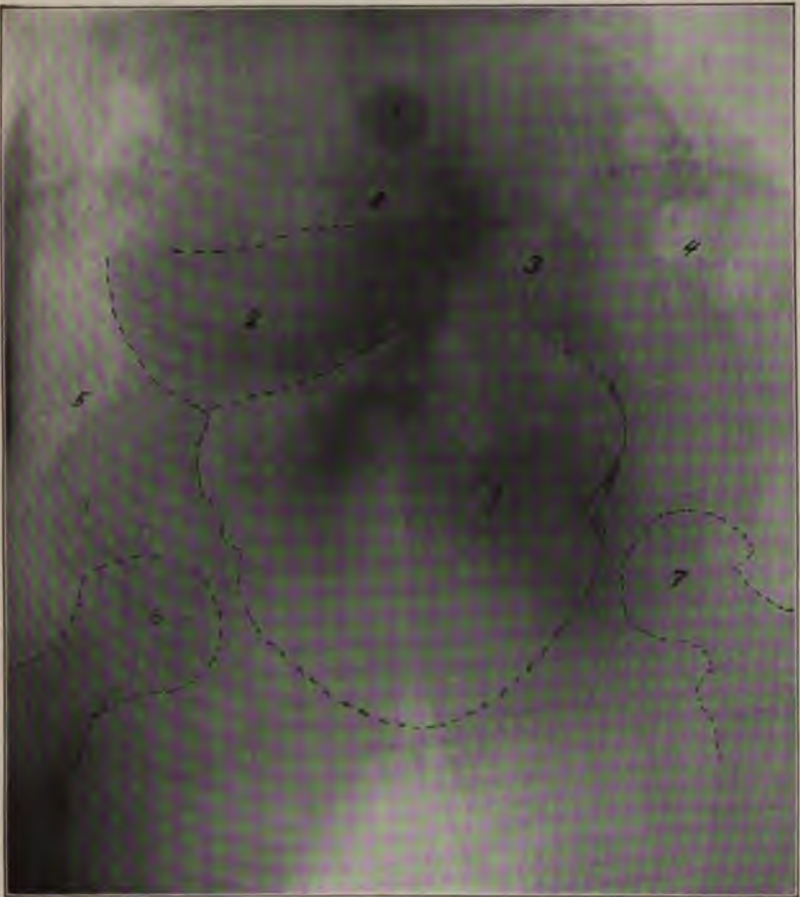
Tortuosity of the Arch of the Aorta Indicated (*A*) by the Aortic Shadow Being Projected Almost Directly Outward on a Level with the Interspace. (*B*) Hypertrophied Left Ventricle.



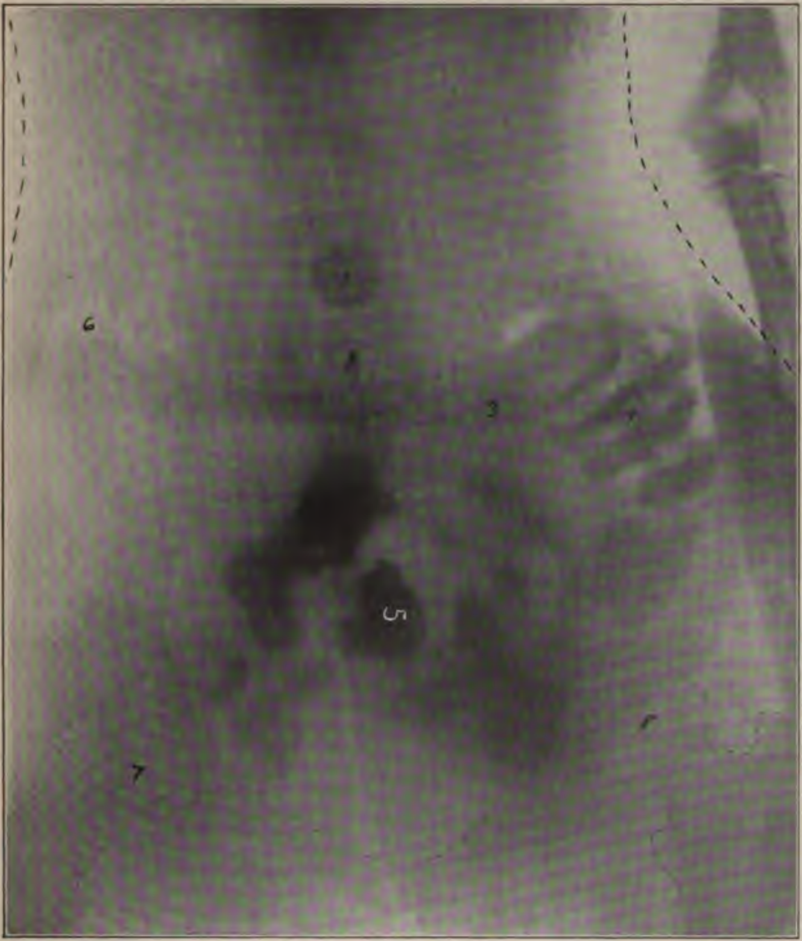
Gastroptosis. (1) One-cent Piece on the Umbilicus. (2) The Stomach Occupying the Vertical Position, and the Pylorus Displaced Downward Four Inches. (3) The Right Sacro-iliac Synchondrosis. (4) The Cecum, Empty. (5) Descending Colon, Also Empty. (6 and 7) Heads of the Femora.



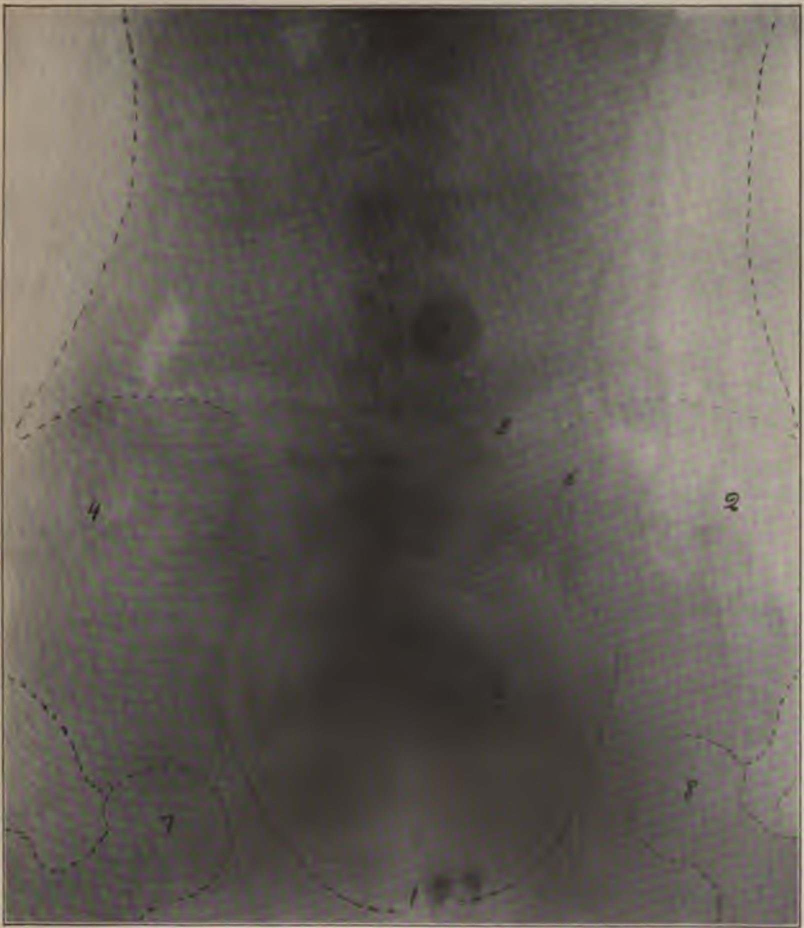
Gastroptosis. (1) A Cent on the Umbilicus. (2) The Stomach, Having Moved Into the Pelvis. (3) Right Sacro-iliac Synchondrosis. (4 and 5) Cecum and Descending Colon, Respectively.



Case of Gastrotopsis. (1) One-cent Piece on the Navel. (2) Stomach. (3) Right Sacro-iliac Synchondrosis. (4 and 5) Empty Cecum and Descending Colon, Respectively. (8) Transverse Colon, Empty. (9) Shadows of Food in the Small Intestine.



(1) One-cent Piece on the Navel. (2) Transverse Colon. (3) Right Sacro-iliac Synchondrosis. (4) Cecum and Ascending Colon, Showing the Food After Twenty-four Hours. (5) Isolated Masses of Food in the Small Intestines. (6) Descending Colon, Empty.



(1) One-cent Piece on the Umbilicus. (2) Cecum and Ascending Colon. (3) Transverse Colon, Empty. (6) Right Sacro-iliac Synchondrosis.

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